THE DIAGNOSTICS OF PAIN

LECTURES FOR MEDICAL STUDENTS AND PHYSICIANS HELD AT THE UNIVERSITY OF COPENHAGEN IN THE AUTUMN OF 1934

BY

TH. B. WERNØE

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LEVIN & MUNKSGAARD
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H. P. HANSENS BOGTR., KBHVN.

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PREFACE.

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Copenhagen, June 1936.

Th. B. Wernøe.

LECTURE I.

Introduction. Sensibility of the tissues. Strength and character, localisation and extension of pain. Classification of pains. Direct pain.

Ladies and Gentlemen.

Pain, as Mackenzie says, is one of the symptoms which most frequently induces the patient to consult a doctor. Sometimes it is the only symptom, and in numerous cases the first symptom, of an affection. Often, therefore, the physician is obliged to base his diagnosis exclusively on the pain present in the case, and consequently it is important for every clinician of any standing to be able to form an expert judgment of such pain phenomena as occur.

Pain, however, of whatever kind it be, is due to an excitation of sensory nerve elements, and must therefore be regarded as a symptom of neurological irritation. For this reason the varied types of pain are most conveniently evaluated from neurological points of view; and after having now for a number of years, aided by a grant from the P. Carl Petersen Foundation, occupied myself partly with clinical, partly with experimental studies on the diagnosis of pain, it occurred to me that I might perhaps be able to give an exposition penetrating more deeply into the facts coming within this field than that found in the text-books usually employed. But before I begin these lectures, I should like to remind you that this is not the first time a Danish clinician has devoted special study to the problems and the diagnostics of pain. Both earlier and more recent Danish clinicians, and indeed other Northern clinicians too, have made important contributions to the

diagnostics of pain. I shall revert later on to several of these works, at present I confine myself to reminding you that Carl Lange, in his General Pathology of the Spinal Cord which was published in 1871—76, was the first to describe thoroughly referred pains from the visceral organs. In 1898 Knud Faber, at the same time as Head and independently of him, published his investigations on visceral reflex hyperaesthesias, and Viggo Christiansen's monograph on migraine appeared in 1925.

Thus, far from indicating a rupture with the Danish medical tradition, my investigations on the problems and the diagnostics of pain must be regarded as continuing the line marked out by the works of the aforementioned investigators.

It results from the nature of the case that the present work has been founded on clinical observations. These observations I have been enabled to make partly during my time as assistant physician at the Neurological Department of the Rigshospital, and partly during my activities as a neurologist at the Finsen Light Institute and, further, as a private practitioner. In addition, I have drawn largely upon the clinical researches of previous investigators, and in my attempt to elucidate obscure mechanisms of pain and my endeavours to find out the laws that govern these mechanisms, I have also utilised experience derived from the experimental investigations which I had the opportunity of making at the zoophysiological laboratory during the years 1923—28 under the guidance of Professor A. Krogh.

Finally, since I began this work I have, partly on myself, partly on patients, studied the mechanism of referred pain by means of electrical stimulation, just as I have made further studies on deep sensibility.

The complete work comprises a general part and a special part. The general part here published deals with the sensibility of the tissues, direct pain and indirect pain (divided into projected pain and referred pain, as well as pain following upon

primary motor excitation, pain arising from primary vegetative excitation, and psychogenic pain), and further combined pains.

Any conceivable pain may presumably be classed under one or the other of these forms.

Sensibility of the tissues.

The first question that arises in connection with the diagnostics of pain is: From what organs and tissues can pain emanate?

Important contributions towards the answering of this question have been given by surgeons who have performed operations under local anaesthesia and have mechanically stimulated the various tissues and organs during the operation. According to the experience of these surgeons, pain caused by mechanical stimulation may emanate not only from the skin, but also from the fasciae, tendons, muscles, periosteum, and bone tissue; and even the capsular ligaments and the synovial membranes are susceptible to mechanical stimulation.

Yet no decisive importance can be ascribed to the information gained by mechanical stimulation.

Thus, those of you who are accustomed to perform lumbar punctures will perhaps have noticed that the patient registers pain when the needle is carried through the skin, whereas it may be carried virtually without pain through the subcutaneous tissue and the muscles, nor does it give rise to appreciable pain if the needle touches the periosteum and the bone tissue. Only at the moment when the needle penetrates the dura is a distinct response observed, and if the needle touches caudal filaments, lancinating pains are felt in the lower extremities.

This, however, does not render possible any general conclusions concerning the strength of the pain which may emanate from the various tissues through which the needle is carried; for upon irritation of another kind, the distribution of pain may be quite different. One may convince oneself of this by means of experiments with Faradic stimulation.

Thus, if a fold of the skin is stimulated by a strong Faradic

current, a severe superficial pain is felt. If the electrode is placed over superficially situated muscles, a severe superficial pain is still felt; further there is a feeling of contraction and perhaps also a faintly marked deep-seated dull pain, though merely inconsiderable. But if the electrode is pressed firmly against superficially situated bone tissue, a violent, deep-seated, dull and gnawing pain is felt, which quite overshadows the pain in the skin.

The pain in the bone becomes still more distinct at the expense of the pain in the skin, if a method employed by me is adopted, which might perhaps be called the displacement method. It is as follows. In a place where easily displaced skin covers superficially situated bone tissue a blunt metal instrument is pressed firmly against the skin and the underlying bone. This produces a pain which must either come from the skin, the periosteum, or the bone, or from the three tissues in conjunction. The pressure is repeated, but first the skin is pushed so much to one side over the bone that the instrument touches another part of the skin, while the pressure comes in the same place on the bone.

The instrument is removed, the skin slips back into place, and the irritated parts of the skin and of the bone now lie far from each other.

I have made most of the experiments on the patella, because the skin covering the edge of it can easily be pushed up over the patella, against the fore edge of which the instrument is pressed. After the compression is discontinued, the skin slips back to the side region, whereafter the patient, with eyes closed, must indicate the seat of the pain. Without exception patients have given the front surface of the patella as the seat of the pain, which would seem to indicate that the pain produced by the pressure of a blunt instrument comes chiefly from the bone or periosteum and not from the skin.

This fact is evidenced even more plainly if the pressure, after a preceding displacement of the skin, is made so strong and prolonged so much that the tenderness lasts for some minutes after it is discontinued.

After the skin has slipped back the patient is told to find the tender spot with his finger. Without exception this is always pointed out as corresponding exactly to the place of compression of the bone, not the place of compression of the skin. Hence it appears from these experiments that the bone tissue and the periosteum are more sensitive to pressure with a blunt instrument than the skin, whereas, conversely, the skin is more sensitive to pin-pricks.

Perhaps the insensibility of the bone to pin-pricks is connected with a fact pointed out already by Gowers who in his lectures issued a warning against the use of pointed needles for sensory examinations, because a pointed needle, in places where a sensory reticulum is found, perhaps may enter one of the meshes without touching the sensory nerve elements themselves. A blunter needle has a greater chance of touching these, and so he recommends the use of a common pen, neither excessively pointed nor excessively broad, instead of a pointed needle.

Perhaps there is, as far as the periosteum and the bone tissue are concerned, a network of nerves into the meshes of which the point of a needle may slip without producing pain, whereas the blunt instrument acts on a larger surface and hence can only with difficulty avoid touching sensory nerve elements, even though these form a relatively wide-meshed net.

In the last instance the sensibility of the tissues is probably dependent on the function of the tissues, since the skin, which serves as a protection against highly varying stimuli of the external kind perhaps for that very reason is sensitive to a series of different stimuli, whereas the deeper-lying tissues are only sensitive to a smaller number of stimuli, and principally those to which the tissue in question is usually exposed owing to its situation and function. The bone tissue, according to its position, structure, and function, will chiefly be exposed to pressure and blows. It is not surprising, therefore, that it is more easily irritated by pressure and blows than by pricks or other external stimuli, which only exceptionally reach the periosteum or the bone.

Muscle tissue, too, is fairly insensitive to pin-pricks whereas vigorous compression of a muscular abdomen will be painful, and if the muscle is the seat of pathological changes, even a slight compression may give rise to intense pain.

Under normal conditions the irritability upon pressure is not, however, very great in most muscles, whereas forced contractions rapidly produce intense muscular pain. For the study of muscular pains I have therefore employed static contractions both on myself and on patients. The subject is, for instance, made to abduce a little finger maximally and continue to keep the finger in this position. Pain will then arise, even though with individual differences, in the course of a few minutes, pain that is felt to be more and more unpleasant the longer the contraction is kept up.

Forced contraction thus furnishes an adequate stimulus for muscular sensory nerve elements, and it is not surprising therefore that muscular cramps and prolonged contractions may be accompanied by severe pain, which is in poor agreement with the conception of muscular sensibility that may be gained by means of mechanical stimulation.

I mention these examples in order to emphasise that each separate tissue has its own peculiar mechanism of pain, so that different tissues responding by severe pain upon adequate stimulation, prove almost insensible when the same stimulus is applied, for instance pin-pricks; in the skin these produce pain, while it is possible to pass the needle through the subcutaneous tissue and the muscles and touch the periosteum and the bone tissue almost painlessly.

Rational as it might then immediately appear to be to investigate the sensibility of the tissues layer by layer by incisions, scratches or pricks during operations, the information to be gained in this way is, however, merely of limited value and does not warrant conclusive inferences with respect to the importance of the various tissues as factors productive of pain.

This is a fact which must be taken into account when we proceed to deal with the tissues which are quite insensitive to mechanical stimulation.

Those of you who have seen a craniotomy performed under local anaesthesia will probably have noticed that the operation is not accompanied by pain except at the moment when the dura is cut through.

After this operations can be performed on the brain without the patient feeling any pain, and only ligatures of larger vessels are accompanied by pain.

Similarly the abdomen may be opened and incisions made in the organs without giving rise to pain. Scratching, burning, and cutting of the organs are likewise painless; according to the investigations of Lennander and Mackenzie the abdominal organs are altogether insensitive to operative incisions, and yet under other circumstances the most acute pain may emanate from the same organs, as known for instance in the form of biliary colic, renal colic, intestinal colic, birth pangs etc.

Here, then, we observe in an intensified form the phenomenon already mentioned. The organs may be insensitive to the ordinary mechanical stimuli, and yet respond by intense pain to an adequate stimulus.

As far as the muscular hollow organs are concerned the adequate stimulus is convulsive muscle contraction or expansion. In this respect I need only remind you of the birth pangs which, as you can easily convince yourselves at births, accompany the contractions of the gravid uterus, and of the pain that accompanies a strong distension of the vesica.

Cardiospasms, pylorospasms, and the spasms associated with enteritis, cholelithiasis, and nephrolithiasis are likewise accompanied by severe pain.

On the other hand, you know also that distension of the stomach and intestine may be accompanied by pain, to which may be added that Poulton and, in Denmark, Stürup in experiments on themselves produced severe pain by distending the esophagus by means of a rubber ball introduced into it.

Whereas the organs themselves seem to be quite insensitive to operative incisions, the case is different for the perivisceral connective tissue. There can hardly be any doubt that those inquirers are right who contend that mechanical stimulation

of this tissue may produce direct pain. Mackenzie, who has made particularly thoroughgoing investigations on the sensibility of the viscera, does not seem to have been sufficiently aware of the difference which may exist between the sensibility of the organs themselves and that of the connective tissue surrounding them; and some of the objections raised to his theories are, indeed, due to this circumstance. I shall subsequently revert to Mackenzie's investigations, but already now I call attention to the fact, not unknown from other parts of the body, that hollow organs vegetatively innervated are less susceptible to irritation than the surrounding connective tissue. You have probably all observed that a solution of salvarsan can be injected without pain into a vein, while soreness and pain arise if merely a single drop is placed outside the vascular wall. Thus the intima is but slightly sensitive to chemical stimuli, and indeed it is likewise insensitive to the mechanical stimulation due to the cannula introduced into the vein. Similarly, the whole vascular wall would seem to be fairly insensitive to electrical stimulation. At any rate, on passing a Faradic electrode over superficially situated large arteries or veins, I have never been able to trace any intensification or merely change in the dermal pain caused by the faradisation. And yet it is a well-known fact that very acute pain may be caused by arteriospasms, and that pain may accompany phlebectasias and varices.

In reality, then, vascular sensibility bears no small resemblance to the sensibility which we find on examining the viscera. This is not to be wondered at, in so far as both the vessels and the viscera are tissues innervated by the vegetative system.

When we add to this the well-known fact that pain may be caused both by irritation of the nerve trunks, the nerve roots, and the central sensory nerve elements, it will be understood that pain may emanate from virtually all the organs and tissues of the body.

In view of the fact that adequate stimuli will give rise to pain of great intensity in almost all the organs and tissues of the body no great diagnostical value can be attached to the intensity of pain, all the more so since great individual differences assert themselves on this point. The same stimulus which will cause intolerable pain in one person, will hardly be heeded by the more robust. To this must be added that insignificant and passing affections often cause great pain, whereas serious organic lesions run an almost painless course. Thus transient arteriospasms may often be accompanied by the most acute pain, while extensive malignant diseases may run a painless course. It may further be mentioned that prolonged subminimal irritation may suddenly by summation set loose violent attacks of pain, and finally it may be added that in pathological conditions the pain receptors may be transformed, so that the threshold for pain is lowered. The result will be that an irritation that does not cause pain under normal circumstances, will be able to evoke impulses which, conducted to the cerebrum in intensified form, there give rise to pains of a severity normally only produced by much stronger stimuli. — All these circumstances render it impossible to base far-reaching diagnostic conclusions on the intensity of pain, apart from the fact that very intense pain localised to the extremities, the neck, or the trunk may with a probability verging on certainty be considered of non-cortical origin, since cortical lesions are only accompanied by paraesthesias or by pain-like paraesthesias, but never by pain of considerable intensity.

The nature of the sensations which may accompany one and the same affection may also vary greatly.

Thus migraine may be accompanied by pain varying from an uncomfortable feeling of heaviness in the head to intense aching. Neuritis may be accompanied by purring, pricking, stinging paraesthesias, by persistent pain or by acute paroxysms

of pain, and similar conditions occur in the case of other forms of pain also.

Hence the nature of the pain may vary in one and the same affection, and information as to this factor cannot, any more than information as to the intensity of the pain, form the basis of far-reaching diagnostic conclusions.

It may be of importance to ascertain whether special circumstances cause exacerbation of the pain; whether it is aggravated by heat or cold, changes of weather or psychic influences; whether the patient feels most comfortable at rest or when moving; whether the pain comes on after meals, after urination or defecation, or is most pronounced in the morning, at noon, in the evening, or at night. All this will be discussed in more detail in the special part of this work under each separate form of pain.

But the most important thing in a diagnostic respect is to obtain exact information as to the localisation and mode of spreading of the pain; for it must be kept in mind that of whatever kind the cause, pain is always in the last instance due to an irritation of sensory nerve elements. Hence pain is to be interpreted as a symptom of neurological irritation, and a rational diagnosis of pain must in consequence be made from neurological points of view. Like all other neurological diagnostics it aims exclusively at determining the site of the underlying lesion, while conclusions as to the nature of the affection must be drawn on the basis of the history and the information obtained through the general medical examination. However, in order to draw conclusions from the localisation and extension of the pain to the site of the underlying lesion it is necessary first to be clear how pain emanating from the different tissues is localised and spread.

Classification.

All forms of pain may, in my opinion, be most conveniently divided into three main groups, viz. a) direct pains, localised to the site of irritation, b) indirect pains, localised at some distance from the site of irritation, and c) combined pains, consisting simultaneously of direct and indirect pains.

a) Direct pains, localised to the site of irritation, are always due to an irritation of the terminal elements of the sensory nerve paths; for if the irritation is of the more centrally situated nerve elements, the pain will, according to the law of the projection of pain, be projected to the peripheral area of the sensory nerve paths, and the pain in question must then be classed under indirect pains.

b) Indirect pains may be divided into 1) projected pains, and 2) referred pains or irradiating pains.

1) Projected pains are due to an irritation of the sensory nerve elements centrally to the peripheral area of extension, and may therefore arise from the nerve trunk, the root, the posterior horn or from sensory elements at a still higher level.
2) Referred pains comprise the indirect pains caused reflexly. To these may further be added
3) Pains arising from primary irritation of the motor system,
4) Pains due to primary irritation of the vegetative system, and
5) Pains dependent on psychic causes.

c) The third main group, the combined pains, is represented by all the cases accompanied simultaneously by direct and indirect pain.

All conceivable pains may presumably without constraint be classed under one or the other of these forms.

As already mentioned, the first proup of pains with which I shall deal comprises direct pains.

It has previously been stated that upon adequate irritation pain may arise from virtually all the tissues and organs of the body; but these pains cannot all be classed under direct pain.

Thus direct pain is not caused by irritation of the vessels or of the central nerve elements; direct pain may, however, be evoked both by mechanical, electrical, and chemical stimulation of the skin, subcutaneous tissue, musculature, tendons and capsular ligaments, periosteum, and bone tissue: further, by irritation of the conjunctiva, the mucous membrane of the nose and mouth, and the pleura parietale, peritoneum parietale, and the subserous tissue, as also of the dura cerebralis and spinalis.

The diagnosis of direct pain must in the first place be based on a careful local examination, and in so far as the pain is due to an external easily demonstrable lesion or affection the diagnosis causes no difficulty, the pain being localised to the site of irritation. The case is otherwise when deep-seated diseases, not easily accessible to the investigator, are involved. But for these pains, too, the diagnosis must be based on a careful local examination. Under this head may be included stethoscopy, exploration, x-ray examination, and in short all the methods used by the clinician for local examination, methods which you will find described in the clinical text-books usually employed, and the more detailed discussion of which does not come within the scope of this work. — I merely wish to mention one or two facts relating to direct pain. Direct pain may occur either as an isolated phenomenon or accompanied by hyperalgesia of the tissue to which the pain is localised. This hyperalgesia, which for the skin may be delimited by means of pin-pricks or folding of the skin, and in the case of the more deep-seated tissues by palpation of the tissue or by exploration, renders it possible for the physician to make an objective examination of the pain indicated by the patient, and often he will be able by a careful local examination to demonstrate a hyperalgesia corresponding to the site of the pain, and so in the case of direct pain to the seat of the irritation.

The hyperalgesias may be unilateral or bilateral, they may be limited to a single tissue or involve several different tissues and sometimes all the tissues, so that hyperalgesias may be termed regional, and according to their localisation certain conclusions may be drawn as to the underlying lesion. This will be more fully discussed in the special part.

But pain may also occur as an isolated factor unaccompanied by hyperalgesia, and the question then arises what inferences may be drawn from the patient's description alone. This will depend on the certainty with which the patient is able to localise his pain — both as far as superficial and deep-seated pains are concerned.

The ability to localise pain has been well investigated for the skin. Normally people are able to localise cutaneous pain with a fair degree of certainty, though the power of localising pain is differently developed for the different areas of the skin, a difference plainly evidenced by the sensory areas pointed out by Weber. For the deeper-lying tissues the displacement method adopted by me, with the employment of a blunt metal instrument after previous displacement of the skin, seems to me well suited. Personally I have used it especially for examining the sensibility of the bone tissue and the periosteum, but I have likewise, after preceding displacement of the skin, tested the sensibility of muscles, tendons, and ligaments by means of a metal instrument with the result that the patients in question have been able to localise the pain caused by pressure with a fair degree of certainty, in so far as they could unfailingly, or almost unfailingly, point to the site of the pain, both when the muscles, tendons and ligaments, and the periosteum and bone tissue were tested. Thus, according to my investigations, patients are able, in the case of deep-seated pain also, to point out the surface localisation of the pain with sufficient certainty for clinical diagnostic purposes, whereas considerably greater uncertainty prevails with respect to the localisation in the deeper strata. Pain caused by irritation of the skin is always unfailingly felt as a superficial pain, but pain emanating from the deeper-lying strata is very doubtfully localised. Thus pain due

to compression of superficial muscles is often appreciated as deep-seated pain, whereas pain evoked by pressure against the periosteum with a metal instrument is as a rule stated to be superficial, or at any rate more superficial than the pain caused by the compression of muscle.

The uncertainty prevalent in the localisation of pain in the deeper strata is amongst other things plainly evidenced by the fact that compression for instance of the pectoralis major causes pain which is stated to be distinctly more deep-seated than the pain evoked by pressure with a metal instrument on the costae lying below the pectoralis major.

I have been able to demonstrate similar conditions in other parts of the body, where the anatomical conditions rendered possible examinations of the above-mentioned kind. Pressure on a bone with a metal instrument will usually cause pain which is felt to be rather superficial, even if the bone is covered by a thick layer of muscles.

Conversely, a deep gnawing pain is felt if the Faradic electrode is placed for instance against a knuckle or against other superficial bone tissue, and a powerful Faradic current passed through the lobe of the ear causes a peculiar deep griping pain. Thus it is not possible to attach any great diagnostic importance to the patient's statements as to the depth of the pain. Pain felt to be deep-seated may come from fairly superficial tissue while pain felt to be more superficial may emanate from deeper-lying tissue.

I emphasise this uncertainty with respect to the localisation of pain in the deeper strata, because it may often give rise to diagnostic errors, if too much weight is attached to the patient's indication of the depth of the pain. Notably it often happens that muscular pain is stated to be deep-seated, as though it were felt right in the middle of the head, the chest, or the abdomen, despite the fact that the underlying muscular lesion can be plainly demonstrated in the muscles surrounding these cavities of the body.

Another subject upon which I should like to enter is the relation between meningeal pains and the pains emanating from central sensory nerve elements. In the text-books meningeal lesions are dealt with under the diseases of the central nervous system. This is only natural in view of the close vicinage of the meninges and the central nervous system, which easily causes meningitical processes to spread to this system; but on the other hand, it must not be left out of consideration that the meninges are innervated by peripheral sensory nerves, and that meningitis pains must be regarded as direct pains of a peripheral kind and not as pains of central origin.

It is true that a meningitis may spread to the central nervous system or to the nerve roots issuing from it, and thus cause pain which is projected to the periphery; but apart from this, meningitical pain is localised to the head and back without showing any special tendency to irridiation of any kind. The pains in the back do, indeed, extend beyond the column on both sides, which would seem to indicate a diffuse localisation, but irradiations to more remote parts of the body are not usually observed, and there can hardly be any doubt that both the pain accompanying cerebral meningitis and that accompanying spinal meningitis must be regarded as direct pain of peripheral origin.

It is of some importance to keep this fact in mind, in the first place because it makes it understandable that persistent pains in the back and persistent headache may be due to a chronic meningitis, even if no symptoms of an affection of the central nervous system are demonstrable; and in the second place because if we maintain that the meningeal pain is a direct pain, a focal diagnosis will often be possible which might otherwise perhaps cause difficulties. If, for instance, a patient only exhibits two symptoms, permanent pain in one side of the head and paraesthesias in the opposite half of the body, it may, if only for this reason, be inferred with preponderant probability that the patient is suffering from an affection which has simultaneously attacked the meninges with consequent direct pain, and the cortex cerebri with resulting crossed paraesthe-

sias. If at the same time there is percussion tenderness corresponding to the site of the pain, this furnishes a further hold in respect of a focal diagnosis.

However, it cannot be expected that a tumor or a localised meningeal process can be delimited on the basis of the distribution of the pain, since, as previously mentioned, the localisation, as far as the meningeal pains are concerned, seems to be rather diffuse. Thus an extensive persistent pain of meningeal origin need not result from a corresponding meningeal process. Beyond the fact that a meningeal process must be assumed to occur within the area of pain, conclusions can hardly be drawn as to the localisation and extent of the underlying meningeal process.

Finally I would like to draw attention to the fact that direct pain may emanate from the nerve trunks. It is indeed a well-known fact that projected pains may be caused by the irritation of nerve trunks without being accompanied by direct pain of any kind. Thus prolonged gentle compression of a nerve trunk is usually unaccompanied by direct pain, but by pains or paraesthesias which are projected to the area of the nerve trunk in question. These pains are due to an irritation of the long paths connected with the trunk. They are felt at a long distance from the site of irritation and thus belong to the indirect pains.

But if brief but strong pressure be exerted on the supraorbital or infraorbital nerve with a finger pulpa, a peculiar local pain is distinctly felt, which is not evoked by pressure on the adjacent parts of the tissue, and no accompanying projected pains or paraesthesias, unless the nerve is exposed to blows or a particularly violent compression. In that case both direct and indirect pain may result, as known for instance from a knock on the elbow. — The direct pain caused by pressure against a nerve stem must in the nature of the case be due to an irritation of peripheral sensory end organs; for pains produced by the irritation of paths central to the peripheral area of distribution are localised, according to the law of the projection of pain, to the peripheral area of the paths in question. The sensory end organs concerned must be connected with the nerve trunk

or rather the nerve sheath, for pressure against the tissue outside the nerve trunk does not cause a corresponding peculiar local pain like that caused when the pressure is on the nerve trunk. The presence of peripheral terminal ramifications in the nerve sheaths has, as a matter of fact, long since been demonstrated (Sappey, Horsley and others), and Gowers mentions in his lectures that the fibrous nerve sheaths are abundantly supplied with nerves which here form peripheral terminal ramifications without penetrating into the interfascicular septa. These ramifications seem, at any rate partly, to originate from neighbouring nerve trunks, since tenderness of the nerve sheath may persist for some time after paralysis of the nerve trunk concerned has set in. Thus the trigeminal trunks must be supposed to receive branches from facialis, since tenderness of the trunks may be present even though the trigeminus root has been severed.

But whether the nerve sheath is innervated by branches from neighbouring trunks alone or by the nerve trunk itself also, it is important, in a diagnostic respect, to keep in mind that peripheral sensory end organs are found in the nerve sheaths, and that irritation of a nerve trunk therefore may involve direct pain emanating from the nerve sheath, in addition to the pain which can be localised to the peripheral area of the nerve trunk and which is due to irritation of the long paths of the nerve trunks. —

Later, when I discuss the pain emanating from the nerve trunk, I shall revert to these questions. But before concluding my remarks on direct pains, I should like to point out that their intensity is not only dependent on the stimulus employed and on the condition of the tissue from which the pain emanates, but also on the condition of the paths by which the impression of pain reaches consciousness. Organic changes of a peripheral or central kind may give rise to an intensification of the impulses conducted from the receptors of pain, and the consequence will be that the threshold for pain will be lowered so that stimuli which are not heeded under normal conditions may

be accompanied by considerable pain. Conversely, destructive affections may involve a weakening of the pain impulses, so that the tissue becomes less sensitive, or insensitive. Cerebral functional disturbances may, however, also entail respectively hyperaesthesia or impaired sensibility, the latter even in so marked a degree that the functional anaesthesias belong to the most pronounced ever met with in clinical medicine. Functional anaesthesia, as I shall show later on in more detail, is due to a functional blocking of the cerebral paths. It may disappear from one day to another and be replaced by its contrary — hyperaesthesia.

Blocking, however, also occurs within the physiological domain. The pressure and friction of clothes against the skin passes unheeded under normal conditions, contractions of the muscles may be performed during a walk without the impulses evoked thereby reaching consciousness, and as an example well suited to illustrate physiological blocking it may be mentioned that normally the heart-beat is not felt, despite the fact that every contraction of the heart entails impacts against the cerebro-spinally innervated wall of the thorax. It is a well-known fact that in nervous patients this anaesthesia may be replaced by a pronounced hyperaesthesia so that normal contractions of the heart cause great discomfort.

On the assumption that a corresponding but merely much deeper-lying blocking occurs in respect of the organs innervated from the vegetative system, it will be understandable that direct pain does not usually emanate from these organs; but at the same time it would indeed be conceivable that neuropathics, as asserted by some enquirers, may sometimes be able directly to localise pain emanating from a bronchial ulcer, an intestinal ulcer, etc. — Both Foerster and v. Bergmann mention examples of this kind. Perhaps, however, it is more probably the perivisceral connective tissue which in these cases has been the startingpoint of the pain. For, as shown by Kappis, direct pain may emanate from this tissue, and precisely an ulcer might easily be supposed to irritate the perivisceral tissue by penetrating

through the wall of the organ and then attacking the surrounding tissue.

This brings to an end my remarks on direct pain. I now turn to indirect pain which is more complicated in a diagnostic respect.

LECTURE II.

Indirect pain. Projected pain: trunk pains, root pains.

Ladies and Gentlemen.

As previously mentioned, indirect pain means pain localised to some part removed from the site of irritation. Hereunder may be classed projected pains, referred pains, and pains due to primary motor and vegetative irritation, as also psychogenic pains. Properly speaking it would be natural at the present point first to deal with referred pains, because these pains, like the direct pains, emanate from peripheral nerve distributions. If nevertheless I prefer to deal first with projected pains, it is because referred pains can more readily be analysed when the rules for the distribution and localisation of projected pains have been elucidated.

Projected pains may emanate from any point of the sensory nerve paths situated centrally to the peripheral terminal distributions. Hence they may emanate both from the nerve trunk and the posterior root, from the posterior horn, the antero-lateral column and the thalamus, and projected paraesthesias, at any rate, may emanate from the cerebral cortex.

A nerve trunk may be affected by a noxa in three ways: either in a purely irritative, or a purely paralytical way, or it may be in part irritated and in part paralytic. Further the trunk may be totally or partially affected, only certain pathways (for instance the sensory ones) being attacked, while the motor paths remain intact.

If we disregard the diffuse forms and those which selectively attack the motor or vegetative paths of the nerve trunk, and consider exclusively the sensory forms, these may then be of

a purely paralytic, of a paralytico-irritative, and of a purely irritative kind.

The purely paralytic kinds are merely accompanied by sensory deficiency symptoms and in those cases the diagnosis must be based exclusively on a demonstration and delimitation of the anaesthesia. In so far as this anaesthesia shows a distinct correspondence to the area of distribution of a certain nerve trunk, the diagnosis may be easy. Often, however, it is difficult, because the distributions of the various nerve trunks partially overlap, so that the anaesthesia produced by paresis of a single nerve trunk may be inconsiderable and too insufficient to base an exact diagnosis on.

Thus sensory paralysis of the radial nerve does not usually give rise to anaesthesia, because the innervation areas of the median and ulnar nerves extend so far over the radialis area that these nerves are in all essentials capable of taking over the innervation if paralysis of the radial nerve sets in.

Occasionally, however, paralysis of the radialis is accompanied by a minor anaesthesia localised to the radial third of the dorsum manus, whereas an anaesthesia corresponding to the whole radialis area is virtually never observed.

If the radial nerve is the site of a paralysing irritative affection, the picture is altered. Anaesthesia in spots is in that case to be regarded as an anaesthesia dolorosa, i. e. an anaesthesia accompanied by pain. The pain is due to irritation of the central part of the interrupted paths and in conformity with the law of the projection of pain it is localised to the peripheral area of distribution of the radial nerve. The pains are not, however, limited to the above-mentioned cutaneous anaesthesia, but spread beyond this into the whole of the radialis region, involving the so-called maximal zone of the nerve in contrast with the anaesthesia which involves the so-called autonomic zone, or particular zone, as I prefer to call it, i. e. the zone in which the radialis appears as the exclusive innervator. (Fig. 1).

In addition to the projected pains the paralysing irritative trunk affection may also be accompanied by local pain in the trunk which, as previously mentioned, emanates from the nerve

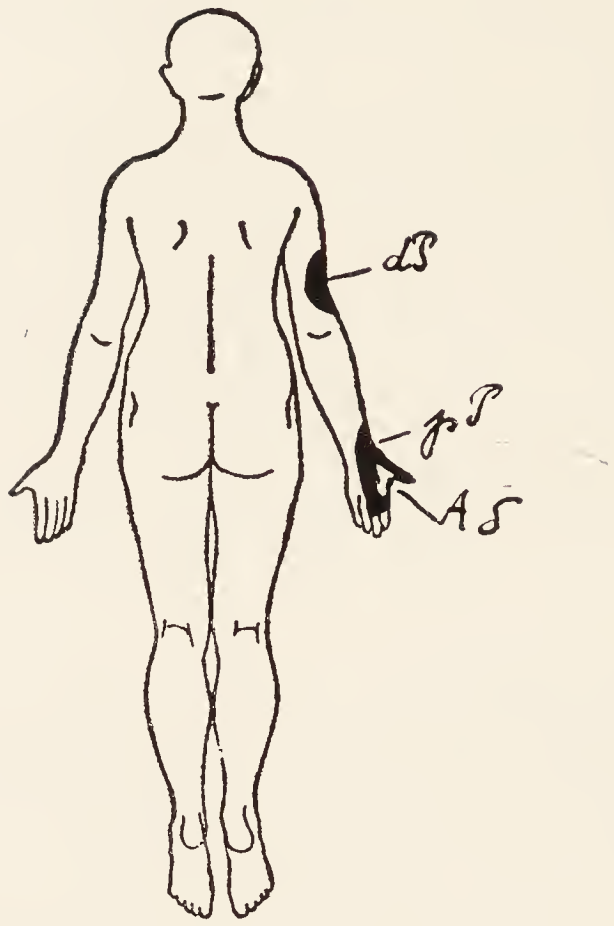

Fig. 1.
Paralytico-irritative affection of the right radial nerve accompanied by direct pain (d. p.) and by pains projected to the maximal zone of the nerve (p. p.), and by anaesthesia dolorosa (A. d.) corresponding to the particular zone of the nerve.

sheath. At the same time the nerve trunk is usually intensely tender upon pressure, whereas a corresponding tenderness is not found in the adjacent tissue. In so far as the nerve sheath is the site of an inflammation-like affection, the local trunk pains may occur spontaneously and without any external cause whatever, and the tenderness upon compression is in these cases comparable with the hyperalgesia which affects other inflamed tissues owing to an alteration in the tone of the pain receptors.

Often, however, local trunk pains are due to exposure of the trunk to abnormal pressure produced by foreign bodies, by callus, or by luxating areas of bones, by exostoses, tumors, or straining cicatrices. Hence, in the case of mononeuritis, that is to say, an affection in a single nerve trunk, the physician must always carefully ascertain whether a local cause can be detected

in the vicinity of the nerve trunk which may expose the nerve to abnormal pressure.

It is not to be wondered at that, for instance, a scar which exerts a direct pressure on a nerve trunk may evoke local pain and local tenderness upon pressure; for it must be remembered that in the first instance the scar presses on the pain receptors of the nerve sheath, the result of which will be local pains: but these pains may become more and more intense, because the sheath at the same time becomes the site of a more and more pronounced hyperalgesia.

Corresponding phenomena are known for the skin. Local pressure from a tight bandage will perhaps at first merely give rise to slight discomfort, but by degrees the tone of the pain receptors is altered on account of the pain, so that the result may be more and more intolerable pains. Similarly the intense tenderness upon pressure which may be found with furuncles is due to abnormal conditions of pressure in the inflamed tissue and not to local infectious neuritis; for if the furuncle is opened and emptied of pus, the tenderness of the tissue disappears almost instantly, which could not happen if it were due to infectious peripheral neuritis.

The tenderness of the nerve trunk which is due, for instance, to a scar which exerts pressure on the nerve trunk is as a rule confined to the site of compression itself. A case serving specially to illustrate this came under my notice at the neurological policlinic of the Rigshospital of Copenhagen. It was an unmarried joiner, aged 20, in whom a cut across the wrist had developed a hypertrophic scar and following upon this a medianus paralysis both of a motor and a sensory kind. The anaesthesia present was accompanied by pain projected to the medianus zone, and in addition there was tenderness upon pressure of the nerve corresponding to the site of the scar. The tenderness of the nerve trunk was, however, very local, since pressure on it above and below the scar was not accompanied by pain, whereas pressure on the scar where it passed the medianus produced intense pain, evidently owing to a

pronounced but narrowly limited hyperalgesia of the sheath caused by the abnormal conditions of pressure.

The pressure, however, also acts on the long pathways running through the nerve sheaths, — sometimes in such a way that interruption of conduction takes place unaccompanied by pain or paraesthesias — hence with a purely paralysing effect — sometimes with a purely irritative effect and causing pains or paraesthesias unaccompanied by anaesthesia, and finally also with a simultaneously paralysing and irritative effect, the result of which, as previously mentioned, will be an anaesthesia dolorosa. In these cases the pain is due to irritation of the interrupted paths above the site of interruption. Here then, we are not, as in the case of the nerve sheath, concerned with an irritation of the pain receptors, but with an irritation of the long paths themselves during their passage from the periphery to the central nervous system. The tenderness upon pressure of the nerve trunk is unconnected with this process of irritation; for pressure upon the nerve trunk causes local pain, while irritation of the long paths will only cause projected pain. On the other hand, it is probable that irritation of the long paths plays an essential part in causing the severe attacks of pain frequently accompanying irritative trunk affections.

However troublesome prolonged local and projected trunk pains may be, nevertheless what patients with this kind of lesions especially fear is the attacks of pain which may occur explosively at longer or shorter intervals in the trunk affections. The pain may increase till it is almost intolerable; during the attack it usually radiates along the nerve to its peripheral area of distribution, even though radiation in the reverse direction may also be observed.

These pains, which as a rule involve the whole area of extension of the nerve trunk, must be assumed to be due to a summation of subminimal stimuli, which at last causes so strong a tension that a discharge takes place; and precisely because the pains spread over the whole area of the nerve, it is reasonable to assume that a summation has taken place in many of the ganglion cells belonging to the nerve trunk, a

summation which must again be assumed to be due to an irritation of many paths. Local irritation of a nerve trunk, however, can only be assumed to involve irritation of a limited number of nerve sheath paths, whereas numerous long paths may be irritated by the local irritation. If only for that reason it must be regarded as probable that it is irritation of the path and not of the sheath which is the cause, or the chief cause, of the attacks.

To this must be added that the pain in the nerve sheaths due to irritation of the pain receptors is usually more intense than the pain caused by irritation of the long pathways. Frequently even a strong irritation of these merely causes paraesthesias instead of pain. It would not, therefore, be improbable that precisely a weak but continual irritation of the long paths might especially be able to bring about the summation of subminimal impulses, the final result of which is the attack of pain.

Why the pains sometimes during the attack radiate along the nerve towards the periphery and sometimes, though more rarely, the opposite way is a question about which various ingenious hypotheses have been put forward. According to Pierret the length of nerve fibres bears a certain relation to the size of the ganglion cells, so that short paths combine with smaller cells and longer paths with larger cells. As a consequence saturation of impulse would occur first in the smaller cells, which would then be discharged first, whereafter discharge of the larger cells would take place, and for that reason pain would first be felt in the area of the short paths and then successively in the area of longer and longer paths. On this hypothesis, which has been put forward by Vanlair, it would be possible to explain descending pain, but it remains to explain ascending pains. True, it has been assumed that these were due to irritation of recurrent paths given off from neighbouring nerves to the nerve sheaths, along which they ascended to higher levels to end, however, before the foramina intervertebralia. The shortest of these recurrent paths would then be found in the periphery and the longest in a more central

situation. On discharge of the corresponding cells the pain would then first be localised to the periphery and successively more and more centrally. It is, however, doubtful whether the nerve sheaths possess recurrent paths of the above-mentioned kind. By electric stimulation of a long nerve trunk, for instance the ulnar nerve, I have at any rate been able to cause local pain, and pain projected to the hand, but no ascending pain, as might have been expected, if the stimulation reached long recurrent paths.

A more simple procedure is, as suggested by Gowers, to parallelise the whole process of discharge with the sensory epileptic aura which may pass now from the arm to the foot, now in the opposite direction.

There is one circumstance, however, which tells against parallelisation of the epileptic sensory aura with the above-mentioned attacks.

The sensory epileptic aura spreads in accordance with the position of the cortical centres, so that the underlying process of irritation must be assumed to spread continuously in one direction or the other from the centre to neighbouring centres. The ganglion cells which belong to a single nerve trunk are not, however, situated side by side but are distributed over several segments. Thus the radialis receives paths both from the fifth, sixth, seventh, and eighth cervical and from the first dorsal segment. The same five segments, according to Spalteholz, likewise give off paths to the median nerve; and the ulnar nerve receives paths from the seventh and eighth cervical and the first dorsal segment. Thus a nerve trunk only occupies part of a segment, but is represented simultaneously in several segments. Therefore it is difficult to understand that a violent sensory irritation should spread from segment to segment and yet only affect precisely those ganglion cells which belong to one definite nerve trunk without irritating the surrounding ones. It is also difficult to imagine that the discharge should be able to take place at the same time in a whole series of spinal ganglia; and a cortical discharge can hardly occur, since a cortical irritation is only accompanied by paraesthesias or at most pain-

like paraesthesias but never by intense pain like that which accompanies the neuralgias. Thus the discharge is probably of a spinal kind, but nothing certain is, in fact, known about the precise mechanism of the discharge. It seems most probable to me, however, that the direction of the pain is dependent on the factor which starts it. If, for instance, we assume that a high-level irritative trunk affection has charged the ganglion cells both of the nerve sheath and the long paths, then a pressure, for instance against the nerve trunk, will first and most strongly irritate the pain receptors of the nerve sheath, the result of which may be a discharge which begins in the corresponding ganglion cells, and which will then, in a way unknown to us but probably in conformity to law, successively involve all the other charged ganglion cells, the result of which will be descending pains.

Conversely, in peripheral neuritic processes, long paths may be irritated more than the above-lying shorter paths, the result of which may be attacks of pain of an ascending type. Thus, for instance, I had under treatment a university lecturer, 37 years old, who at the first examination presented symptoms of a high sciatic neuritis of a paralytico-irritative type. The patient exhibited local pain, high-seated tenderness of the nerve trunk and muscles, loss of the ankle jerk, positive Lasègues symptom, and cutaneous hyperaesthesia in the sciatic zone of the foot, but no tenderness of muscles or nerve trunks below the upper third of femur; slight diminution of vigour in active flexion and extension of the foot, further, no motor pareses. The patient at this point complained partly of permanent local high-seated pain, partly of fits of pain of the descending type. Massage treatment was recommended, but the paient consulted me again after a fortnight on account of pains. This time, however, the pains were localised to the inner side of the knee and the lower part of the femur, and there were fits of pain radiating from here towards the hip, i. e. pain of the ascending type. Both the semitendinosus and the semimembranosus were now intensely tender upon pressure, and pressure against the underlying bone tissue, too, was accompanied by pain. Presum-

ably, then, during the treatment with massage, descending neuritic processes have arisen, which have reached the above-mentioned peripheral tissues, whereafter attacks of pain of the ascending type have arisen.

Several similar cases could be mentioned, and even though fixed rules cannot be established, I am, nevertheless, of opinion that neuralgic attacks of the ascending type as a rule, or always, upon closer examination will prove to be due to a peripheral process of irritation.

Often vasomotor phenomena accompany the fits of pain, phenomena which Gowers, however, merely regards as secondary and without direct bearing on the mechanism of the attacks. For the redness and burning of the skin which so often accompanies the attacks frequently occur before the pain. Thus this sequence could be very distinctly observed in a married woman, 51 years old, in whom the ramus superficialis of the left radial nerve had been injured in an operation (extirpation of a lipoma). The patient had for about a year before the operation been suffering from pains which radiated from the elbow down along the radial side of the left forearm into the dorsal side of the thumb and index finger. At the same time there was just below the elbow a swelling which upon operation turned out to be caused by a lipoma the size of a walnut, through which the ramus superficialis of the radial nerve passed. The tumor was removed, the wound healed uneventfully and the pain disappeared; but attacks of pain reappeared fairly quickly after operation. The pain radiated from the scar towards the thumb and index finger. Corresponding to the first metacarpal interstice was found an about 2×3 cm sized cutaneous anaesthesia as a symptom of a sensory radialis paralysis and otherwise no signs of organic nerve affection. Each attack of pain, however, was preceded by reddening of the skin above the scar and only after this did the pain occur. A hyperaemia at the site of irritation would thus in this case seem to be the factor that started, or at any rate preceded, the attacks.

Hence the sensory symptoms that accompany the paralytic-irritative trunk affections are these:

1) Anaesthesia, co-extensive with the autonomic zone of the nerve; 2) projected pain, often in fits, corresponding to the maximal zone of the nerve trunk; 3) local trunk pains, and 4) tenderness upon pressure of the nerve trunks.

In the purely irritative forms there are likewise pains corresponding to the maximal zone of the nerve trunk; at the same time cutaneous hyperalgesia in the peripheral area of the nerve trunk and tenderness of the corresponding muscles will usually be found. Tendons and ligaments, too, may be tender upon pressure, and occasionally pressure against bone tissue may likewise be accompanied by pronounced sensations of pain.

Hyperalgesia of the area of the nerve trunk may be observed in high-seated irritative trunk affections, and in those cases is probably due to the circumstance that impulses arising from peripheral irritation are intensified during their passage through the ganglion cells already irritated.

But the hyperalgesia may also be due to low-seated neuritic processes, in which case it is much more pronounced, probably owing to a change of tone in the pain receptors. When pronounced tenderness of the muscles in the peripheral area accompanies a trunk affection, it is thus usually a case of a low-seated mononeuritis or else a polyneuritic affection. In contrast with mononeuritis, which is as a rule due to a local cause, polyneuritis is caused by circulating toxic substances which attack the peripheral nerve elements, for which they have a special affinity. It is principally the distal part of the nerves of the long extremities which are attacked, and whether of the paralysing or the irritative form, the nervous affection is bilateral and symmetrical. Probably because the affection shows a predilection for the distal branches of the nerves, polyneuritis is often accompanied by much tenderness of the muscles. Thus it is a well-known fact that alcoholic polyneuritis and arsenical polyneuritis are accompanied by pronounced muscular tenderness. Great tenderness of the muscles may likewise be observed in influenza neuritis and in other forms of infectious neuritis, and the rarer arteriosclerotic polyneuritis has, at any rate in

the cases that have come under my observation, been accompanied by an intense tenderness of the muscles.

Tenderness of the nerve trunk and muscles, pain localised to the maximal zone of the nerve trunk, and hyperalgesia with the same delimitation are thus the chief symptoms of the purely irritative trunk affection.

With all forms of trunk affections there are, however, often associated secondary symptoms of a motor or vegetative kind. These are especially pronounced in the trigeminus affections which are often accompanied by twitching of the facial muscles, tic douloreux, reddening and hotness of the skin of the face and perspiration and hypersecretion of tears. Similar symptoms may occur in affections of the medianus and other trunk lesions. This was already mentioned above and will be treated in more detail in the section dealing with pain arising as a secondary symptom in primary irritation of the vegetative nervous system.

If now we proceed to consider the pain due to root affections, it must again be emphasised that an affection of a nerve-root may be either 1) purely paralysing or 2) simultaneously paralysing and irritative or 3) purely irritative.

Paralysis of a single nerve-root causes no demonstrable anaesthesia, because the segments overlap, so that adjacent segments can take over the lost innervation. If, however, paralysis of several neighbouring roots arises, this paralysis is accompanied by a radicular or segmental anaesthesia.

Not only on anatomical maps, but also in most text-books on neurology you will find figures with the segmental boundaries marked and perhaps you have noticed that the diagrams in question are hardly ever alike. This is scarcely because one enquirer has made less careful investigations than another, but the reason is that the cutaneous boundaries of the segments may vary. Variation with respect to limitation of segments may be distinctly observed in the skin of the fish, whose pigment

cells are innervated sympathetically. If the sympathetic is severed between two ganglia, the skin pales upon stimulation of the sympathetic above the place of section but grows darker below the place of section owing to paralysis of the pigment cells, the so-called chromatophores. This will cause a strong contrast between the light and the dark area of the skin, and the boundary line corresponds to the lower limit of the segment lying above the place of section.

When preparing my thesis for the doctorate, I made numerous experiments of this kind and found that the segments are not bounded by straight transverse lines, but the boundary line winds irregularly in and out, so that it must be kept in mind that even in these markedly segmented animals the segmental boundaries will vary. It is therefore the less to be expected that constant boundaries can be found in man whose structure is far less segmental than that of the above-mentioned experimental animals.

But apart from these variations the course of the segmental boundaries is so characteristic that a segmental anaesthesia, when present, can usually be diagnosed without difficulty and be used as a basis for conclusions with respect to the level of the underlying affection.

In addition to purely paralytic root affections there may, however, also occur paralytico-irritative forms of root affections, but only in so far as the spinal ganglion is entirely or partly preserved. In the opposite case ascending degeneration will set in, which will render possible the incidence of root pains.

If the affection only comprises a single root, it will not, however, as previously mentioned, be possible to demonstrate anaesthesia.

On the other hand, in paralytico-irritative forms pains will in all cases be able to assert themselves.

It is true that root affections are often solely accompanied by paraesthesias in the form of tingling, crawling, pricking and jabbing sensations. The patients in question may have the feeling of walking on cotton, they may have girdle sensations

and other special sensations, such as often accompany tabetic root affections. But what is especially characteristic of root irritation is, however, pains which may partly occur in fits as lightning stabbing and lacerating pains (the so-called lancinating pains) and partly as more rheumatism-like pains of a more stable and fixed character, which spread segmentally and thus do not follow the course of the nerve trunk, but extend like bands down the extremities; they are not localised to the peripheral area of the nerve trunks, but to peripheral segmental zones. Since root affections are most frequently due to spondylitic or meningeal affections, as also to extra-medullary tumors, the posterior roots are often attacked bilaterally, so that doublesided root pains often occur, causing the so-called girdle pains on the chest and abdomen and bilateral lancinating pains in the extremities.

Girdle pains as well as bilateral pains in the extremities must always arouse suspicion of the presence of root affections.

As previously mentioned, radicular anaesthesia dolorosa is accompanied by pain with a segmental or radicular projection, and both girdle pains and bilateral pains of the extremities may occur in conjunction with anaesthesia of radicular origin. Lancinating pains, too, may accompany the paralytic anaesthetic root affections.

It is, hovewer, especially in the incipient stage that a patient with tabes dorsalis is troubled with lancinating pains, which can evidently only arise as long as sensibility is partially preserved. For during the attack, and often for some time after the attack, there is usually at the site of the pain a pronounced cutaneous hyperalgesia, so intense that even the lightest touch, even merely the pressure of the clothes, may cause considerable pain; but such a hyperalgesia cannot, in the nature of the case, arise if all paths from the periphery to the central nervous system were interrupted. The presence of intact peripheral sensory nerve paths thus seems to be a prerequisite for a root irritation to cause lancinating pain. The most natural explanation of this seems to me to be that an antidromic effect asserts itself with respect to the lancinating pains.

Irritation of the peripheral part of a divided posterior root may, as shown by Bayliss, Doi, Krogh, Harrop, and Rehberg, cause vascular dilatation. To all appearance this is due to impulses caused by the irritation, which are conducted backwards along the peripheral sensory paths to the skin, with a resulting vascular dilatation. Thus impulses may be conducted backwards along peripheral sensory nerve paths, and Foerster has, as a matter of fact, in human subjects, both by electric stimulation of the peripheral stump of severed nerve trunks and by electric stimulation of the peripheral stump of severed nerve-roots been able to evoke pain in the area of distribution of the nerve trunks and nerve roots concerned. Thus stimulation of the peripheral stump of the severed cutaneous surae lateralis was accompanied by pain in the peripheral area of the nerve. Anastomoses must accordingly be assumed to occur between the terminal branches of the cutaneus surae lateralis and the cutaneus surae medialis, and by an antidromic effect, that is to say by sensory impulses conducted backwards, the stimulation must be conducted through anastomoses to the terminal ramifications of the cutaneus medialis; for after an ensuing severing of this nerve, pain could be evoked neither by stimulation of the peripheral part of the cutaneus medialis nor of the cutaneus lateralis.

Similarly Foerster has stimulated the peripheral part of a severed posterior root and produced pain in the zone of the root concerned. Only after severing both of the neighbouring roots could pain no longer be evoked by electric stimulation.

Accordingly, on stimulation of the root in human subjects, impulses may be conducted backwards along the peripheral sensory paths and so evoke pain, and since lancinating pain only seems to arise by irritation of roots whose peripheral paths have been preserved, it would be natural to suppose that these pains were due to an antidromic effect of the above-mentioned kind. I have ascertained by experiment how rapidly an antidromic effect may occur on fish skin. If, after a preceding decapitation and destruction of the cord, one of the posterior

spinal roots is stimulated with a needle, a dark zone corresponding to the root zone concerned is produced with lightning rapidity, dilatation of the pigment cells setting in. This effect cannot have been produced along vegetative paths; for however sensitive to vegetative stimulation the fish skin may be, the effect is always produced relatively slowly (i. e. in the course of about 3—5 seconds), whether evoked by the stimulation of central or peripheral vegetative paths. On stimulation of the posterior root, on the other hand, the antidromic effect is, as has already been mentioned, produced with lightning rapidity.

Similarly, in a human subject, irritation of a posterior root will probably be able to evoke impulses which will shoot with lightning rapidity into the area of the root, where they will reach the pain receptors belonging to the neighbouring segments through anastomoses. But in that case this peripheral irritation will at the same moment evoke pain which according to the nature of the case the patient feels as the stab of a lance, a dagger, or a knife. — If the lancinating pains, as is usually the case, are accompanied by hyperalgesia, all the sensory connections between the peripheral and the central nervous system cannot be interrupted, and it is indeed a well-known clinical observation that the lancinating pains in tabes chiefly occur in the incipient stage, the so-called neuralgic stage, which as the first stage precedes the eventually ensuing second (atactic) stage and the third (paralytic) stage; I take it as evidence that lancinating pains arise by the above-mentioned peripheral irritation evoked by an antidromic effect, that the patient's response to them occasionally has the character of a parrying action.

An intelligent tabetic once said to me: »The pain was in fact felt as if it were the stab of a dagger, and instinctively I thrust my hand towards the place to seize the dagger.«

Patients suffering from malignant trigeminal neuralgia do not try to remove the irritant, but press their hand against the affected cheek to allay the pain.

LECTURE III.

Continuation. Root pains, pains from the posterior horn, pains from the antero-lateral column.

Ladies and Gentlemen.

Besides lancinating pains tabes dorsalis is also often accompanied by fits of pain in the organs — the so-called crises. These pains, however, do not occur as lightning pains, but whether in gastric or intestinal crises, cardial, vesicular, renal, or bladder crises, the pain is felt as if it emanated from the organ in question. Hence it is not surprising that these pains have in the course of time given rise of many erroneous diagnoses, the crises having been regarded as signs of serious diseases in the organs in question.

Already the demonstration of corset aneasthesia or other segmental anaesthesias, or information as to preceding lancinating pain or girdle pains will, however, always excite suspicion of the presence of a tabetic affection, and a systematic neurological examination should always be made in these cases before the final diagnosis is given.

If tabetic crises occurred as a constant symptom in irritative root affections it would be natural to co-ordinate these pains with the pains which otherwise accompany irritative or paralytico-irritative root affections, as is indeed often done in the literature. Crises, however, virtually occur exclusively in tabes dorsalis, but not, on the other hand, in similar wise in root affections caused by spondylitic or pachymeningitic affections or in such as are due to extramedullary tumors. From this I think it possible to infer that there is an additional irritative factor in tabes which does not occur in the above-mentioned

irritative root affections. Pathologically and anatomically, besides the root lesion and meningeal changes, there occurs in tabes in the first place degeneration of the posterior columns, a degeneration which does not occur in a corresponding way in spondylitic lesions, and it would therefore be natural to regard an irritation caused by the degeneration of the posterior column as the cause of the crisis. It is especially the paths serving the muscle sense which pass to the posterior column, and thus probably also the afferent fibres of the smooth musculature. The paths run directly from the root into the posterior column, where they are continued in long ascending pathways which connect with nuclei in Goll's and Burdach's columns at the beginning of the medulla, and in short descending paths which, as stated by Strümpell, are in all probability reflex paths. Accordingly irritation of the posterior column may probably through these paths irritate vegetative spinal ganglion cells and thereby cause contraction of smooth muscles, and with the tendency of the central nervous system to sum up irritative impulses so as finally to bring about a violent discharge, it would be understandable if the crises were introduced by a spinal vegetative discharge with resulting convulsive contractions of the smooth muscles of the organs and with ensuing pain. The contractions would then in the tabetic crises be a tertiary pain factor, whereas the spinal vegetative discharge would represent the secondary pain factor, and the irritation of the posterior column the primary element.

According to Foerster there occurs in the great majority of gastric crises a marked cutaneous hyperalgesia from the 5th to the 9th dorsal segment or still lower; Foerster supposes this to be due to irritation of the roots issuing from afferent visceral paths.

However, this view is hardly correct. For if, owing to the presence of disturbances of conduction during gastric crises, impulses from afferent visceral paths could be transferred directly to the cutaneous paths of the root, then it might also be expected that, conversely, the impulses might be transferred from irritated cutaneous paths to the visceral paths. In other

words, this would mean that lancinating pains should always or frequently be accompanied by crisis pains, which is not the case.

On the contrary, crises virtually never occur simultaneously with lancinating pain. This is easily understandable in so far as it is a well known clinical and experimental fact that painful stimulation reflexly exerts an inhibiting effect on visceral contractions. As a consequence, the lancinating pains will in all probability, while they last, suppress rather than produce crises.

The primary irritation which causes the crises must therefore be assumed to take place in a part where the afferent visceral paths are separated from the pain paths. The pain paths run from the posterior root to the posterior horn, whereas the visceral paths in all probability run from the posterior root to the posterior column like other visceral muscular paths. It is then reasonable to suppose that the irritation which causes the crises emanates from the posterior column and not from the posterior root. —

The hyperalgesia which accompanies for instance gastric crises is hardly, either, to be considered as a result of a root irritation. A similar hyperalgesia very often accompanies visceral lesions of quite another origin and, as will be explained later in more detail, they are in all probability to be regarded as a reflex phenomenon.

You might perhaps ask whether it were not possible that peripheral disturbances caused the spasms accompanying the gastric and other visceral crises.

The reply to this is that apart from the fact that the crises are probably due to irritation of the posterior column, while the lancinating pains must be assumed to be due to a root affection, the two forms of pain are otherwise comparable. They both occur in tabetics in fits of extreme violence and of shorter or longer duration, but before and after the fits the patient is quite well, a fact which is only with difficulty reconcilable with the assumption that peripheral gastric disturbances should be the cause of the crises.

There can hardly be any doubt that the gastric crises are

due to a discharge of a central kind, and in all probability this is again due to a primary irritation of the posterior column.

Before leaving this important section of the diagnostics of pain, I shall briefly report, as an illustration of the varied symptoms which lesions of the posterior root may present, some descriptions of symptoms in tabetics derived from Dr. Viggo Christiansen's lectures of 1905. The full description will be found there. I shall confine myself to mentioning the sensory irritation phenomena.

1) A coachmaker, aged 33, declares that his pains come in fits, often lasting several days running, and with a distinct tendency to exacerbation in the night. The pain is extremely intense sometimes forcing him to moan loudly, it is further fugacious, and capriciously localised, now to the thigh, now to the calf, now to the heel or the sole and the toes, boring and tearing as if a knife were stuck into the painful spot. But even though these attacks of pain last for days on end, it is the rule that each single pain is of short duration, only lasting for one or two seconds, to be repeated at shorter or longer intervals during which there is only a burning or smouldering sensation. During the attack of pain, and often persisting some time after its cessation, there is in the area in which the pain is located, a limited but very intense hyperalgesia. Even the lightest touch causes considerable pain, so that the pressure of the clothes or the blanket becomes intolerable to the patient.

In addition to these lancinating pains, which are chiefly localised to various parts of the lower extremities, the patient complains of very intense pain in the urethra, likewise in fits. Both by their mode of occurrence and their character they are highly reminiscent of the lancinating pains occurring in the trunk and extremities of tabetics. As already mentioned, these urethral crises occur by fits, and come on suddenly, lasting for some hours, sometimes a couple of days, to disappear again as quickly as they came.

The patient further complains of a, usually painful, but sometimes merely constrictive sensation round the lower part of the thorax. But besides these, at any rate at times, painful

sensations, the patient also suffers from a swarm of paraesthesias localised especially to the lower extremities. There is partly spontaneous tingling and »pins and needles« in the feet and toes, and partly the very unpleasant feeling when he puts his feet to the ground that he is walking on felt. In the upper extremities too, and especially in the right limb, he has paraesthesias which, as is the rule in tabes, are localised to the ulnar fingers.

2) The patient, a man aged 41, began 6—7 years ago to have attacks of pain in the abdomen; this pain was extremely severe and is so still. The onset is quite sudden, and the pain is so violent that the big man screams and moans loudly, writhes and bites his handkerchief. They are accompanied by severe vomiting and a prostration approaching collapse. The patient's appearance is collapsed, his skin pale, white, cool, the pulse weak, and he feels extremely exhausted. After the attack has lasted for a couple of hours it disappears again as suddenly as it came, and between the attacks the patient feels perfectly well. The frequency of the attacks is very variable, but their paroxysmal occurrence has struck the patient himself. As a rule they come for some days in succession, almost only occurring in the morning, and he is then spared for some months. During the paroxysms the abdomen, despite the severe pain, is quite natural and soft, not even tender. As long as the paroxysm lasts there is complete anorexia, but between the fits there are no gastric symptoms of any kind.

3) The third patient complained that the day before she had quite suddenly been attacked by intolerable pains in the abdomen, accompanied by a strong constrictive sensation of the abdomen in conjunction with incessant vomiting. Her skin was cold and perspiring, her pulse weak, and her expression was somewhat like that which accompanies serious abdominal cases. She was quite unable to eat anything, every attempt to make her drink merely caused increased vomiting. The abdomen was perfectly natural, not distended, not contracted, there was some tenderness on the left side, but is was slight and unaccompanied by the *defense musculaire* so characteristic

of peritoneal irritation. On being questioned, the patient explained that for the last five years she had suffered from similar attacks. They occurred regularly at intervals of 2—3 months and a couple of years before she had been treated in a surgical department for them. The onset was always quite sudden, they generally lasted for 3—4 days, and disappeared as quickly as they began.

4) The patient is a man, 46 years old. 8 years ago he began to have lancinating and lacerating pains, now in one now in another place, though there was a predilection for the tip of the left shoulder-blade, for the posterior and anterior surface of the thigh, and for the calves. These pains, which are extraordinarily severe, so that loud moans are forced from the patient, come in well defined fits, which may last from some hours to several days. Each fit consists of a series of pains lasting from some minutes to half an hour, and each single pain lasts only for some seconds. After the pain has raged for some time in one place, a pronounced hyperalgesia sets in here, so that the contact of his clothes, or in the night of the blanket, is quite intolerable to him.

5) The patient is a man, aged 45. About a year ago he began to have unpleasant sensations around the thorax and the upper part of the abdomen; varying somewhat in intensity and extension, they might spread from the claviculae to the horizontal through the upper part of the iliac crest, at other times they were confined to a girdle at the level of the third and fourth costae. There was no actual paroxysmal occurrence of these constrictive sensations, they were present almost constantly, only more or less pronounced, there has never been actual pain any more then crisis-like symptoms.

6) The patient is a woman, aged 50. During a stay in the surgical ward, she experienced the first onset of the pains which have since troubled her. These pains are especially localised to the area below the left shoulder-blade, now and then they also occur below the right one, she has a sensation as if a knife was stuck into the sick place. The pains occur in fits, radiate into the chest and last from one to several

days. They are extremely intense, but each single pain is brief. The attacks begin and stop quite suddenly.

Thus, in the first patient there occurred lancinating pains, urethral crises, girdle sensations, and various paraesthesias, while the second and third patients suffered from gastric crises. In the fourth patient there were lancinating pains, in the fifth patient accentuated girdle sensation, and finally in the last patient, lancinating pains.

(Besides the above-mentioned symptoms, the history of the patients in question showed lues, Argyll-Robertson's symptom, ataxia, lost knee- and ankle jerks, extensive anaesthesias or other tabetic symptoms, which rendered the diagnosis conclusive).

From the descriptions you will, however, presumably have gained a vivid impression of the varied multiplicity of paraesthesias which may be a result of tabetic lesions, and at the same time of the strength with which tabetic pains may occur. Another symptom of interest is the flaccidity of the abdominal wall which could be demonstrated during the attacks in two of the patients suffering from gastric crises. The tension of the abdominal wall which accompanies severe visceral pain is due, according to the researches of Mackenzie and of Sherrington, to a spinal motor reflex and the loss of it in the two cases described must be assumed to be due to a degeneration of the posterior column and is thus comparable to the tabetic's loss of the knee and ankle jerks. For, the severe pain would seem to indicate that the paths of the posterior root are intact, and motor paralyses did not accompany the cases described.

While, as previously mentioned, irritative lesions of the nerve trunks and of the posterior roots are very often accompanied by pains which at times may reach the highest degrees and approximate to the intolerable, pain does not belong to the symptoms characteristic of the medullary lesions.

Extensive intramedullary tumors may develop without having been accompanied by pain, and myelitic processes may run a painless course. It is, however, not rare to see pain caused by intramedullary lesions.

Thus the central gliomatous process accompanying syringomyelia has frequently an irritative effect on the ganglial cells of the posterior horn, before the destruction takes place. Similarly, both haematomyelia and intramedullary tumors may have an irritative effect on the posterior horn cells with resulting severe pain.

Occasionally the pain accompanying syringomyelia and intramedullary tumors is due to a simultaneous affection of the meninges and posterior roots; but in other cases it was not possible, on section, to find any sign of a lesion of the meninges or the roots, so that it must be considered beyond doubt that the pain emanated from the irritated posterior horn.

When a complete destruction of the posterior horn cells has taken place, the pain ceases, and it is indeed a well-known fact that pain, for instance in cases of syringomyelia, principally occurs in the incipient stage, whereas pain rarely accompanies the later stages. But at the time when an irritative process occurs, pain may occur, often accompanied by a marked hyperalgesia.

The sensory dificiency symptoms due to lesions of the posterior horn are of a dissociated kind, pain sense and temperature sense being lost, whereas deep sensibility is preserved.

The cause of this dissociation is that all or virtually all pain and temperature paths pass to the posterior horn, from which they cross to the antero-lateral column of the opposite side. A number of tactile paths probably take the same course as the pain and temperature paths. For it has been shown that in the so-called Brown-Séquard paralysis which occurs after a unilateral lesion of the cord, there is not only lost pain and temperature sense on the opposite side but also some loss of the sense of touch, while on the side of the lesion there occurs motor and vasomotor hemiplegia as also loss of deep sensibility, in addition to radicular anaesthesia corresponding

to the site of the lesion; but the reduction of the sense of touch on the crossed side is not very considerable, so that a considerable number of the tactile paths must be assumed to pass the posterior column together with the paths representing deep sensibility.

Thus a lesion of the posterior horn is chiefly accompanied by thermoanaesthesia and by analgesia, and if the whole of the posterior horn has been destroyed, the analgesia in question will as a matter of course be segmental, because all the pain paths to the segment in question will be broken off.

However, in a considerable number of cases of syringomyelia and other central medullary affections there is no segmental analgesia, whereas there is glove analgesia, cuff analgesia, and altogether circular analgesias of the type often accompanying functional lesions. This fact has been erroneously interpreted as a sign that syringomyelia is often preceded by functional symptoms, a view you will find stated in several text-books.

Brouwer, however, has explained this phenomenon in the following way.

A segment of the spinal cord has only a slight depth and bears most resemblance to a flat disc. As a consequence the single parts of a skin segment are not represented by groups of ganglion cells situated at different levels, but side by side. Now Brouwer assumes that the medial, anterior parts of the posterior horn are connected with the distal part of the skin segment in question, whereas the lateral and posterior parts of the posterior horn are connected with the proximal part of the skin segment. As a consequence a longitudinal process which touches for instance the medial anterior part of the 7th and 8th segments of the cord, will cause glove analgesia, whereas a process touching the posterior lateral part of the segments of the arms may cause a circular analgesia of more proximal areas of the arm.

Assuming this explanation to be correct, it will also be understandable that irritative longitudinal medullary processes may be accompanied by regional pains, while irritative proces-

ses with a larger superficial area may involve pains with a segmental distribution.

Anaesthesia dolorosa does not accompany lesions of the posterior horns; for since the point of attack is the ganglion cells themselves, the pain will cease with the incidence of analgesia, owing to destruction of the irritated ganglion cells.

But even though anaesthesia dolorosa in the strict sense does not accompany lesions of the posterior horns, still syringomyelia may very well be accompanied by analgesia and pain at the same time; only the analgesia and the pain are not localised to the same place, though within the same cutaneous segment, since one group of cells in the posterior horn may be destroyed, while another group is still in the irritative stage. In this way, as the gliomatous process progresses step by step, pain and hyperalgesia may be superseded by analgesia.

A case especially suited to illustrate this has been communicated by Foerster, who, during observation for several years of a patient suffering from syringomyelia, noted the following symptoms.

First there developed an atrophic paralysis of the small muscles of the hand on the right side, also an analgesia and a thermoanaesthesia of the fifth to the first dorsal cutaneous segment. The patient complained of excruciating pains in the little, ring, and middle fingers, whereas the thumb and index finger were completely free from pain. Hyperalgesia was present in the ulnar part of the hand as also in the little, ring, and middle fingers, (i e. corresponding to the 8th cervical segment). Gradually the pains ceased, whereafter analgesia and thermoanaesthesia of the ulnar part of the hand and of the ring and little fingers set in. Almost at the same time the pain travelled to the index finger and partly to the thumb, still persisting in the middle finger. Further the patient stated that he often had pains in the middle of the forearm. These pains, too, gradually subsided, analgesia and thermoanaesthesia at the same time spreading over the whole of the hand with the exception of the thenar and the

thumb, and further to the forearm with the exception of a narrow radial stripe.

After this the pains in the thumb became very acute; to a less extent pains were felt in the index finger and up the radial edge of the forearm and the outer side of the arm. In these areas there was pronounced hyperalgesia. In the course of three years the whole hand, the whole forearm, and the greater part of the arm had become analgetic and thermo-anaesthetic with the exception of the upper region of the outer side of the arm. The pains were now localised to this area and to the shoulder, and here there was likewise pronounced hyperalgesia.

The case exemplifies how a gliomatous process may progress from segment to segment, irritating and then destroying, and accompanied respectively by pain and hyperalgesia, and then by anaesthesia and thermoanaesthesia. In this case the process spread successively through the 8th, 7th and 6th up to the 5th servical segment.

The main features of pain from the posterior horn are herewith at the same time indicated. Immediately after its cessation this pain is superseded by analgesia and thermo-anaesthesia. In the case in question the distribution of the pain was segmental, in other cases it may be regional. Personally I have seen several cases of pains in the shoulder region which had been regarded as rheumatism, until the incidence of analgesia and thermoanaesthesia with the simultaneous preservation of deep sensibility revealed the true nature of the lesion. —

After having left the posterior horn the pains rapidly cross over to the lateral column on the opposite side, continuing in this in company with all the other pain paths from the opposite half of the body. The pain paths here seem to be separated from the temperature paths, since there are reports of lesions of the antero-lateral column which have entailed crossed analgesia alone, or crossed thermoanaesthesia alone.

The most frequent clinical observation is, however, that

the symptoms from the anterior column enter as a component into Brown-Séquard's syndrome, which arises from hemilateral lesions of the medulla.

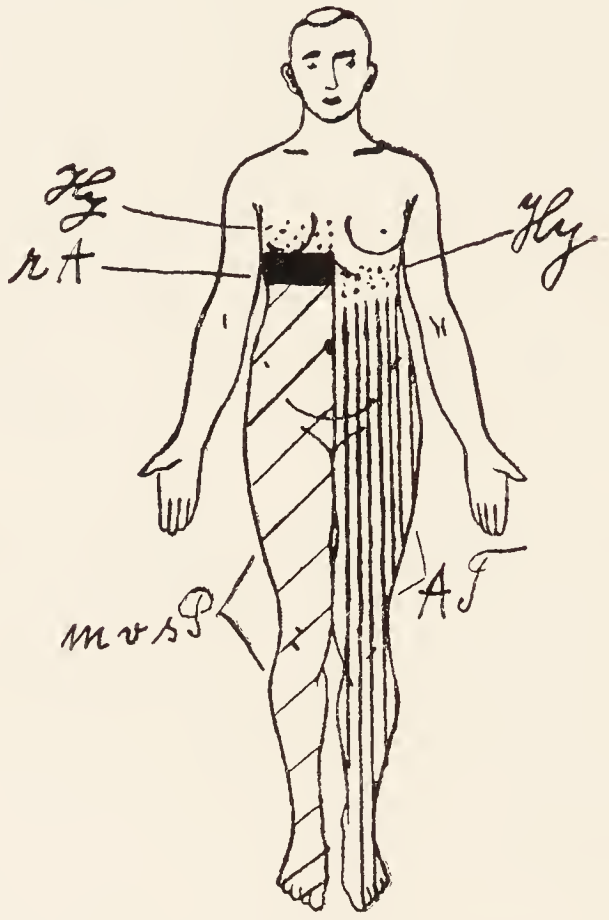

Fig. 2.
Hemilateral spinal lesion entailing on the same side: radicular anaesthesia (rA), also motor and vasomotor hemiparesis and lost deep sensibility (mvsP). On the opposite side: analgesia and thermoanaesthesia (AT). Upwards on both sides: a narrow hyperaesthetic zone (Hy) (Brown-Séquard's syndrome).

As previously mentioned, hemilateral lesions give rise to the following symptoms: On the side of the lesion motor and vasomotor hemiplegia and loss of deep sensibility, further segmental anaesthesia for all qualities of feeling corresponding to the injured segment. On the opposite side there occurs analgesia and thermoanaesthesia which involve the entire half of the body up to one or a couple of segments below the site of the lesion, further there is occasionally blunting of the sense of touch. Finally, above the site of the lesion and above the decussated analgesia there arises a narrow hyperaesthetic zone probably due to an irritative effect of the lesion on the roots and marrow respectively. (Fig. 2).

Hence, in irritative cases one might expect to find, on the decussated side, hemilateral pains as a result of irritation of the anterolateral column. In affections involving pronounced motor hemiplegia on the same side, only sensory deficiency symptoms on the opposite side can as a rule be expected, but in lesions only causing a slight diminution of vigour on the injured side the patient often complains of pains, which may be very severe, or of paraesthesias, on the opposite side.

In hemilateral lesions above the arm segments pain may occur both in the opposite upper and lower extremities and thus be reminiscent of thalamus pains.

If the site of the lesion is below the arm segments, pains may be evoked in the lower extremity on the opposite side and may be mistaken for neuralgias.

These pains, however, differ from neuralgias or trunk pains by the fact that they are not localised to the area of any definite nerve trunk, further there is no tenderness of musculature and nerve trunks, and the upward boundary is segmental.

The hemiplegic form of pain differs from thalamic pains by the fact that it is never accompanied by anaesthesia, while the chief symptom accompanying a unilateral lesion of the thalamus is hemianaesthesia dolorosa localised to the opposite side, and the reason why you never find crossed anaesthesia dolorosa in lesions of the anterolateral column is that destruction of the paths in the anterolateral column entails degeneration of the ascending kind, because the trophic centres of the neurones in question are in the posterior horn. Like the pains from the posterior roots, therefore, the pains from the antero-lateral column also cease as soon as analgesia sets in, on account of the interruption of the paths in question.

Diagnostically it is important to remember that irritative hemilateral lesions may give rise to pains or paraesthesias in the opposite extremity, not only in the case of irritative unilateral lesions, but also when the investigator is confronted with a fully developed paraparesis. It is important to become clear as to whether the pareses of the lower extremities have developed at the same time, or whether the paraparesis started

with hemilateral symptoms. In these cases the physician has only the history to go by, and sometimes the patient states that numbness of one lower extremity set in at the same time as paralysis of the other. But patients, at any rate according to my experience, as a rule remember better whether crossed pains or paraesthesias have been present at the time when the feeling of weakness began in the lower extremity on the affected side, than they recollect a previous crossed anaesthesia or hyperaesthesia. I recommend, therefore, that whenever you are confronted with a case of paraparesis inf. of uncertain origin, you should always ask the patient whether paralysis began in both extremities at the same time, or whether it appeared first in one extremity and then in the other, and whether in that case crossed pains and paraesthesias accompanied the last paresis, and finally whether irradiating pains of the chest or abdomen have occurred.

If the first paresis was accompanied by root pains in the form of irradiating pains in the chest or abdomen on the affected side, and by crossed pains and paraesthesias, this indicates the presence of a hemilateral lesion, most frequently a tumor at the same time affecting the posterior root, the lateral column, and the antero-lateral column. If paresis also sets in on the other side, this is an indication that the tumor has either spread to the other side and directly invaded the sound half of the cord, or that the tumor has increased in volume and thus by causing increased pressure injured the opposite half of the cord. If the paresis that set in last is accompanied by root pains alone, this indicates the presence of a posteriorly located tumor. If crossed pains alone accompany the last paresis it is an indication of the anterior localisation of the tumor.

Diagnostically the above-mentioned combinations of pain are not conclusive. It cannot be excluded that, for instance, a tumor developing in the left posterior root may exert a distant effect on the antero-lateral column on the opposite side, before symptoms are evoked from the other posterior root or from the antero-lateral column on the same side; but usually

it will be possible, by careful questioning concerning the sequence of the pareses and pains, and the localisation and extension of the pains, to acquire information which enables the physician to draw conclusions with a considerable degree of probability concerning the situation of the tumor and the direction of its growth.

I cannot, therefore, fail to advise you, whenever you are confronted by a case of paraparesis inf., carefully to elicit information concerning the sequence of the symptoms, not only when there is already a well-founded suspicion of the presence of a tumor, or when the diagnosis is entirely doubtful, but also when you are confronted with apparently safe diagnoses (lues cerebrospinalis, disseminated sclerosis or the like). Even if the diagnosis is correct, the paraparesis might still be due to a tumor which might possibly with the great progress made in our day in neuro-surgery, be removed with a good result.

It must be kept in mind, however, that however conformable to law the onset of the symptoms would seem to have been, a diagnosis of tumor cannot be made on this basis alone. It must be based on the total objective findings, under this head comes examination of the cerebrospinal fluid, and possibly x-ray examination after injection of lipiodol.

Even the most exact information concerning the sequence of the symptoms can only render it predominantly probable that a localised process is present in the spinal canal, which directly or indirectly influences first one and then the other half of the cord.

Whether this pathological process is an inflammation, a growing tumor, or a process of sclerosis spreading transversally cannot be decided on the basis of the pain alone.

Before concluding my discussion of the pains of the antero-lateral column, I would point out once more that a complete hemianaesthesia dolorosa cannot be due to a lesion of the antero-lateral column; for, simultaneously with the destruction of the paths in the antero-lateral column and the onset of anaesthesia, the pains cease automatically owing to the incidence of ascending degeneration of the paths in the antero-lateral column.

LECTURE IV.

Bulbar pains, pontine pains, thalamic pains.
Cortical paraesthesias.

Ladies and Gentlemen.

The pain paths in the antero-lateral column pass through the cord without interruption, and in the case of some of the paths continue through the brain-stem to the thalamus.

Others terminate in the formatio reticularis and run through the grey matter in short chains of neurones until these, too, reach the thalamus.

The area of the pain paths is thus larger and more diffuse in the brain-stem than in the cord, for which reason localised unilateral lesions of the brain-stem less frequently give rise to pronounced anaesthesia of the opposite half of the body.

Irritative lesions, however, often cause pains or paraesthesias in the contralateral half of the body. The crossed pains in question are usually accompanied by a light hyperaesthesia, probably due to a partial destruction of the rather diffusely located paths. If pains and paraesthesias occur both in the upper and the lower extremity, it may be concluded from the localisation alone that the pains cannot be due to a deep-seated lesion of the antero-lateral column. It should be noted, however, that the diagnosis of pains emanating from the paths in the antero-lateral columns must chiefly be based on the accompanying symptoms. If motor pareses are present the main stress will be laid on them, and the diagnosis of pain will in that case be of less importance. Bulbar lesions are thus frequently accompanied by dysarthria and dysphagia and atrophy of the tongue, which is usually bilateral. In unilateral

affections, however, dysarthria and dysphagia will be but slightly developed or entirely absent. In that case a hemilateral atrophy of the tongue may be confounded with the atrophy due to hypoglossal paresis. Crossed pareses of a hemiplegic kind will in this case be able to reveal the central origin of the atrophy of the tongue. If no motor hemiplegia is present, the presence of crossed sensory hemilateral symptoms will point in the same direction, whether it is a case of hemilateral analgesia, hemilateral pain, or merely hemilateral paraesthesias.

Usually, however, the bulbar lesions can be diagnosed exclusively on the basis of the motor pareses and the accompanying atrophy, and similarly the pontine affections are most frequently recognised solely by the alternating pareses by which they are accompanied. Because the central motor paths running to the cranial nerves cross over to the opposite side already in the pons, there to connect with the nuclei from which the peripheral paths arise, a hemilateral affection of the pons may give rise to paresis of a cranial nerve on the affected side and hemiplegia on the opposite side, i. e. to hemiplegia alterna. Whereas the hemiplegic pareses will be of a spastic supranuclear kind, the paresis of the cranial nerve will be nuclear, inert and atrophic, because the lesion is of the place of origin of the peripheral motor paths of the cranial nerve, and according as the hemiplegia occurs in combination with paresis of the oculomotor, trochlear, trigeminus, abducing, or facial nerves, conclusions may be drawn concerning the seat of the lesion in the pons, for the cranial nerves issue from the brain stem in the above-mentioned sequence. Thus paresis of the oculomotorius with crossed hemiplegia is indicative of a unilateral affection of the anterior part of the pons, whereas paresis of the abducens combined with crossed hemiplegia suggests a unilateral affection of the posterior part of the pons; between these lie affections causing paresis of the trochlearis and trigeminus with crossed hemiplegia.

However, there also occur irritative motor forms giving rise to the syndrome described by Brissaud, viz. tonic or clonic

cramps of the facial muscles and simultaneous motor hemiplegia on the opposite side.

Similarly, if the pontine sensory nerve elements are selectively affected, this may give rise to a unilateral trigeminal anaesthesia accompanied by anaesthesia of the opposite half

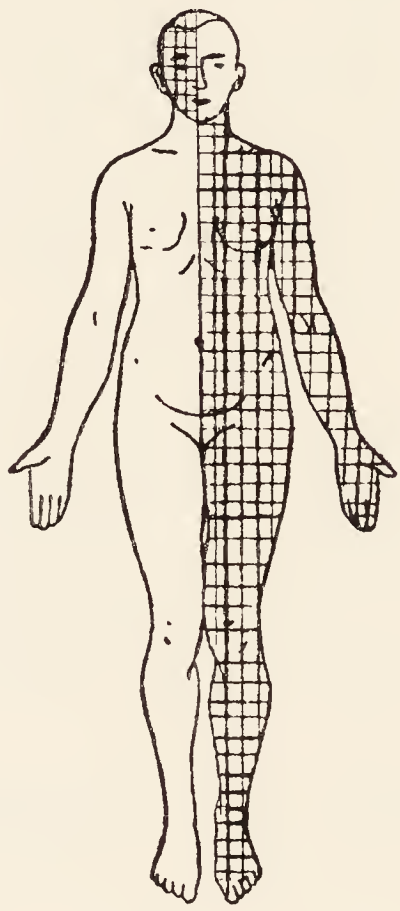

Fig. 3.
Anaesthesia alterna.

of the body, i. e. an anaesthesia alterna (fig. 3). — And in irritative forms unilateral facial pains may be accompanied by pains or paraesthesias in the opposite half of the body. The symptoms may, however, be combined in another way, unilateral motor cranial nerve symptoms may be combined with crossed hemilateral sensory symptoms, and conversely, sensory cranial nerve symptoms may be combined with crossed hemilateral motor symptoms.

Altogether, in lesions of the pons ,there is a possibility of many combinations of symptoms. Thus, for instance, an affection of the trigiminus nuclei may give rise to either sensory or motor or combined sensory motor symptoms. The sensory symptoms may be of a paralytic or of an irritative kind, the motor symptoms may likewise be either of a paralytic or an

irritative kind. Consequently there is the possibility that simultaneous sensory-motor symptoms may occur in the following different combinations: sensory paralytic and motor paralytic; sensory paralytic and motor irritative; sensory irritative and motor paralytic; sensory irritative and motor irritative.

Thus already an affection of the trigeminus nuclei may, even apart from its degree and strength, give rise to a considerable number of different symptoms.

If the pyramidal tracts or the long sensory paths are simultaneously attacked irritatively or paralytically, then again each of the said trigeminal symptoms may be conbined with crossed hemiplegic symptoms which may occur either as sensory or motor or sensory-motor symptoms of a paralytic or an irritative kind.

It will thus appear from this single example alone that unilateral affections of the pons may be accompanied by alternating symptoms in highly varying combinations.

These symptoms, however, may be so rudimentally developed on one or the other side that to the detriment of the diagnosis they may be completely overlooked. As an example I shall exhibit a couple of cases which came under my notice at the Finsen Light Institute and the Rigshospital respectively.

In the first case the alternating hemiplegic symptoms were present in a distinctly appreciable form, whereas the cranial nerve symptoms could only be ascertained through the history.

The patient was an unmarried woman, aged 49, suffering from a spastic paresis of both lower extremities, and a lighter paresis of the left arm unaccompanied by sensory deficiency symptoms. The affection had been diagnosed partly as disseminated sclerosis, partly as a myotrophic lateral sclerosis. At the outset, however, there were certain circumstances which told against both these diagnoses. In the first place the pareses had all on their appearance been accompanied by paraesthesias in the extremities in question. This at any rate excluded the diagnosis of amyotrophic lateral sclerosis, which selectively attacks motor nerve elements.

A diagnosis of disseminated sclerosis was more probable. In the first place the scleroses had set in at two different periods, the patient having developed paresis of the left lower extremity and in slighter degree of the left upper extremity 15 years ago. Only seven years later did paresis of the right lower extremity set in. Further, already on the first occasion her hands had become so unsteady that she could only with difficulty do needlework. These symptoms would naturally suggest disseminated sclerosis.

There were nevertheless, in the development of the disease, facts which told against this diagnosis.

In the first place the patient stated that on both occasions the pareses had followed upon a febrile period, of which she could only say that it had been rather protracted the first time. She had then got up three times but each time had been obliged to go to bed again owing to a return of the fever. When she could finally get up for good, she had noticed that she walked with difficulty. She could indeed stand and walk about by holding on to the furniture; but nevertheless she often fell, not because her legs failed her, but because she missed the objects aimed at, or rather because she aimed beside them. Hence an uncertainty of cerebellar origin. And as a matter of fact, the physician who had treated her stated that at that time she suffered from pronounced dysmetry.

These particulars would seem to indicate that the pareses of the patient were due to an infectious lesion which had at the same time attacked cerebellar paths and paths to the extremities. Hence an infectious lesion of the brain-stem must most probably be the cause of the pareses. As previously mentioned, paresis of the right lower extremity set in seven years later after a febrile period. At the same time the left leg grew strongly paretic, which would seem to indicate that there was no new infectious lesion but a recrudescence of the old one. Crossing the mid-line it had at the same time invaded both the motor and the sensory paths to the right lower extremity, respectively paralytically and irritatively, with resulting motor pareses and paraesthesias.

This diagnosis was so far supported by the fact that examination of the eyes showed nothing abnormal beyond reduced convergence.

Examination of the cerebrospinal fluid showed normal figures ($^2/_3$ cells, 1 globuline, 10 albumine). X-ray examination showed no abnormal conditions. Wassermann reaction negative both in blood and cerebrospinal fluid. Still alternating pareses were not demonstrable.

Further questioning of the patient elicited the fact that since the appearance of the pareses she had often suffered from headache. This was contantly localised to the back of the neck and was accompanied by giddiness and nausea, but not by scintillation. Moreover, the patient had in the course of years had frequent attacks of facial pains which were always localised to the right cheek and spread to the ear, where pains circled round the ear without spreading further backward or radiating in other directions. The pain is thus fixed. It occurs in the trigeminus zone but can hardly be due to a trunk or root affection since it is not coextensive with a single trigeminus branch or with the whole trigeminus area, but only with part of the area of the second branch and at the same time with an area adjacent to and yet outside the trigeminus region. Pains thus localised must be assumed to be of central, and to all appearance of pontine, origin; for cortical lesions are virtually never accompanied by pains, but only by paraesthesias, and thalamic lesions are as a rule accompanied by pains of quite another type.

On the predominantly probable assumption that the pain in question is of pontine origin, the findings in this case are the alternating symptoms characteristic of pontine affections, namely, a lesion of a cranial nerve combined with a crossed hemiplegia, even though the cranial nerve affection is not of a paralysing but exclusively of an irritative kind, and a diagnosis of pontine encephalitis will be even more probable.

In the case in question, however, the diagnosis was not verified by operation, and thus there is no conclusive evidence for the correctness of these inferences.

In another case, referred for examination from one of the surgical departments of the Rigshospital, the circumstances were different. The patient had been operated on before the examination.

This patient was an arteriosclerotic woman, aged 77, who was subject to violent fits of right-sided facial pains, despite the fact that before admission she had had the corresponding trigeminus root resected.

The examination, in addition to right-sided trigeminal anaesthesia, only revealed two symptoms of the presence of an organic nerve affection, namely, reduced vigour of the dorsal reflexors of the left foot and positive Babinski toe phenomenon on the left foot. Hence there was no completely crossed hemiplegia, yet crossed symptoms which, taken in conjunction with the fits of pain in the trigeminus area, must render a diagnosis of pontine affection predominantly probable. As previously mentioned, the pains had been quite unaffected by the resection of the root.

The case shows that the diagnosis of neuralgia n. trigemini should not be made until pontine affections can be excluded with predominant probability. If pronounced alternating pareses are present, a diagnosis of pontine affection may be easy, but often it is difficult, because only rudimentary hemiplegic symptoms occur, the influence and appreciation of which requires great practice and experience of neurological examination. If only for that reason major operations on the trigeminus should never be performed unless indicated by a previous expert neurological examination.

From the brain-stem the sensory paths reach the thalamus where they are all interrupted, to be connected with ganglion cells which form the point of departure for the sensory neurones which conduct the sensory impulses through the semioval centre to the cerebral cortex.

An essential part of the sensory paths, however, hardly

reach the cortex. It must be assumed that part of them are connected reflexly with subcortical motor and secretory centres which serve face play and the secretion of tears, for disturbances in the functions of tears and laughter often accompany lesions of the thalamus.

Further, unilateral lesions of the thalamus are often accompanied by unilateral athetosis and chorea, which would seem to indicate a loss of the regulating impulses to the motor centres in question.

Again, unilateral lesions of the thalamus are accompanied by crossed hemilateral anaesthesia, which is of a dissociated kind, since deep sensibility is affected much more than the pain, temperature, and tactile senses.

It might perhaps be immediately inferred from this that the thalamus plays an important role in the service of deep sensibility. Even if this is correct, the conclusion is unjustified. For provided the interruption of the path be complete, this special loss of deep sensibility only shows that deep sensibility is less bilaterally represented than the pain, temperature, and tactile senses.

But if we add to this the fact that destructive cortical lesions are unaccompanied by a corresponding loss of deep sensibility, there can hardly be any doubt that the loss of deep sensibility in thalamus lesions is connected with the thalamus and not with the cortex cerebri.

It is a well-known fact that complicated processes may take place in deep sensibility without reaching consciousness; not only sleepwalking but also walking in the waking state are in all essentials reflex actions without the direct cooperation of consciousness, but in clinical examinations for the purpose of finding out whether deep sensibility is normal or lost, investigation of the reflexes is not the method adopted but the patient's consciousness is tested. Thus if the patient is asked whether the hallux is bent or stretched, this is in reality a test to find out whether the impulses evoked by the bending have reached the patient's consciousness, and since it is the ability to appreciate changes of posture which is lost in lesions of the

thalamus, but retained in cortical lesions, it follows that the thalamus must possess elements which are capable of appreciating deep sensibility independently.

I premise these remarks before I proceed to discuss the chief symptom in the thalamic symdrome first described by Roussy and Dejerine, i. e. the hemianaesthesia dolorosa which very often occurs in the opposite half of the body in unilateral lesions of the thalamus.

The pains accompanying the thalamic anaesthesia are among the most acute and most persistent ever known. As a rule they are deepseated, often burning; they are permanent, but may increase in fits till they are almost intolerable, and are then unaffected by any medicamental therapy.

It is not difficult to diagnose thalamus pains if complete hemianaesthesia dolorosa is present, for no other nervous lesion is accompanied by this symptom — cortical lesions are rarely accompanied by pain and never by anaesthesia of the type occurring in lesions of the thalamus. And a complete hemianaesthesia cannot accompany lesions of the central nervous system below the pontine trigeminus nuclei, for in that case the hemianaesthesia would not be complete; nor lesions on a level with the trigeminus nuclei, for in that case alternating anaesthesia would occur. On the other hand, a unilateral interruption of all the sensory paths between the trigeminus nucleus and the thalamus might perhaps give rise to complete hemianaesthesia, but then it would probably be unaccompanied by persistent pain, because the interrupted paths degenerate upwards. Hysteria, on the other hand, may be accompanied by pronounced hemianaesthesia, but then deep sensibility is not usually attacked, while the pain, temperature, and tactile senses are relatively well preserved.

It is considerably more difficult to form a diagnosis when there is merely a less extensive anaesthesia. The diagnosis must then be based on the peculiar character of the anaesthesia. In the first place there will as a rule be an anaesthesia dolorosa, and in the second place an anaesthesia with loss of deep sensibility and less reduced pain, temperature, and tactile sense.

In doubtful cases it must further be considered whether the paychic reflexes are reduced, whether there is unilateral athetosis or chorea, and further whether signs can be found of a lesion of the pyramidal tracts, which are easily injured in thalamic affections owing to the near neighbourship.

If none of these latter signs can be demonstrated, and if there are only hints of anaesthesia, it will be difficult to diagnose thalamic pains, though it will always create a suspicion of a thalamus affection, if pain of doubtful origin, especially in elderly arteriosclerotic individuals, proves strikingly refractory to all therapy, for, as previously mentioned, this is a characteristic of pains caused by thalamus affections.

But even if the clinical diagnosis of thalamic pains may cause great difficulty, still greater difficulties are encountered when we attempt to elucidate the mechanism of the thalamic pains.

This is most conveniently done by comparing the symptoms accompanying thalamic lesions with those accompanying cortical lesions. For this reason the mechanism of the thalamic pains will not be discussed in more detail until after the discussion of the sensory symptoms which may accompany cortical lesions.

As previously mentioned, the cerebral cortex is insensitive to mechanical stimulation. The cortex may be cut, pricked, stabbed, and burned without the patient feeling any pain. Not even electrical stimulation of the cortex has as a rule evoked pain, but only tingling, and pricking paraesthesias projected to the extremities or the trunk.

Foerster, however, states that he has evoked pain by stimulation of the parietal lobe and the posterior part of the lobus paracentralis. But apart from this, he, too, could only produce varied projected paraesthesias by electrical stimulation.

Clinical experience likewise shows that cortical lesions are only accompanied by paraesthesias and not by pain, apart from the fact that for instance a compact tumor may evoke pain by pressure on the meninges. However, as already stated, these

pains are direct pains, caused by the irritation of peripheral sensory end organs, and have no connection with the projected sensations produced by irritation of the cortex.

Paraesthesias, however, may also accompany trunk and root affections and central nervous lesions situated below the level of the cortex. The cortical paraesthesias are distinguished from these by their tendency to spread in accordance with the position of the cortical centres, as is especially the case in patients with sensory epilepsy. The paraesthesias may move both upwards and downwards.

If the paraesthesias begin in the foot, the rule is for them to spread from the foot up the leg and further up to the shoulder and then along the arm to the hand and the fingers; they may continue up the neck to the face, where they usually stop at the mouth or the nose.

If they begin in the hand, they pass first to the shoulder, and thence down to the foot. Often they then spread to the other foot and extend upwards on the other side.

In view of the fact that the cortical centres of the foot lie uppermost and immediately at the mid-line, it is understandable that an irritation here relatively easily spreads from one side to the other. With regard to the projected paraesthesias this corresponds to the fact that paraesthesias which have reached one foot, suddenly appear in the foot of the other side.

It is worth noticing, however, that even such a violent cortical discharge as that which must be assumed to take place during sensory epileptic fits, is never accompanied by actual pain, but at most by pain-like paraesthesias.

Fixed paraesthesias, too, may accompany, for instance, a cortical tumor, and in that case the diagnosis may be difficult. But sooner or later motor monoplegia will then be demonstrable. Indeed, fixed cortical paraesthesia differs from trunk paraesthesia in that it does not, like this, respect the limits of the area of the trunk, just as, in contrast to root paraesthesias, it does not respect the segmental boundaries.

Because cortical lesions are unaccompanied by pain, and most investigators have only been able to evoke paraesthesias and not pain by electrical stimulation of the cortex, there are, indeed, observers who, like Head and Holmes, assume that the pain paths only reach the thalamus, not the cortex.

On this assumption the pain-percipient elements should be situated in the thalamus, which would furnish a natural explanation of the fact that lesions of the thalamus are accompanied by severe pain.

Foerster, however, is inclined to refer the perception of pain to the cortex and is of opinion that the reason why thalamic pains may attain so great an intensity is that in thalamus lesions an inhibition of a cortico-thalamic kind has been lost, so that thalamic ganglion cells may uninhibited send impulses to the cortex, with resulting severe pains.

Personally I am of opinion that the perception of pain in man may normally take place both in the cerebral cortex and in the thalamus.

The observations which form the basis of this view are due to investigations made by means of bimanual faradisation, a method which I have previously described, and which will be mentioned in more detail in the special part of this work in the section on psychogenic pains, where I shall revert to the problem of the mechanism of thalamic pains. The theory therefore must for the present remain unproved. But already now I should like to mention a single point which it seems to me should be brought to the front in a discussion on the mechanism of thalamic pain.

The characteristic feature of thalamic pain is not that it is associated with anaesthesia; for anaesthesia dolorosa also occurs in other nervous lesions, for instance in affections of the trunk. Nor is it the tendency of thalamic pain to spread over large areas; for cortical irritation, too, shows a tendency to spread and the processes of discharge which cause the neuralgic fit reveal the same tendency.

The characteristic feature of thalamic pain, on the other hand, is its resistance to the usual anodynes. The cause can

hardly be that the thalamic pains exceed all others in severity, for other pains, too, may increase till they are intolerable. In my opinion the cause is to be found in the fact that in patients with thalamic lesions the anodynes are bound to the pain-percipient ganglion cells less perfectly than usual.

Haemorrhages in the thalamus, which are the most frequent cause of the occurrence of the thalamus syndrome, will inevitably bring about disturbances of the circulation in the organ, which may perhaps result in an irritation of the thalamic ganglion cells and at the same time prevent anodynes from being carried in sufficient amount to the irritated cells with the circulation. In that case it must be regarded as probable that the thalamic pain is felt in the thalamus itself, for if it was felt by the cortex, it must be assumed to be capable of being influenced by the usual anodynes, whereas it is understandable that these are inactive if circulatory disturbances prevent them from reaching the pain-percipient ganglion cells. — On this theory, then, the thalamic pains are felt in the thalamus itself; but it does not follow that all pain is felt by the thalamus, for that the cortex is also under normal circumstances capable of perceiving pain will presumably appear from the fact that cortical lesions are often accompanied by a reduced sense of pain, in spite of the fact that the pain sense, as already mentioned, is bilateral. —

LECTURE V.

Referred pains, visceral reflex hyperaesthesias, vicsero-cutaneous anemic zones.

Ladies and Gentlemen.

The first part of the present lecture is an address previously delivered in the Danish Society for Collective Medical Research, but not published before. I shall give it without alterations in the same concentrated form as then, and merely add some supplementary remarks.

Though referred pains, as pointed out especially by Mackenzie, may accompany both visceral and extra-visceral lesions, it is especially referred pains emanating from the viscera to which investigators have in the course of time devoted attention.

Carl Lange was the first to institute a systematic investigation of these visceral phenomena of pain, subjecting them to a thorough examination already at the beginning of the seventies in his Pathology of the Spinal Cord.

Lange points out that the pains arising in diseases of the internal organs are chiefly referred pains. In his opinion they are caused by impulses conducted to the cord, whence they spread to the sensory elements whose peripheral terminal elements are found in the abdominal wall, and consciousness then projects the pains to this spot.

Lange likewise observed that there is often cutaneous hyperaesthesia in the painful spot, and he points out the great

importance of establishing the laws or chief rules, if possible, in conformity to which the irradiation takes place.

About 20 years later Mackenzie, Head, and Faber independently of each other published their well-known works on the visceral referred pains and reflex hyperaesthesias. While Head and Faber studied the hyperaesthetic zones with special thoroughness, Mackenzie soon took a wider view of the whole subject, which he made clear partly in a series of shorter studies published in 1892—1906, partly in his »Symptoms and their Interpretation« published in 1909.

Mackenzie regards all visceral pains as referred pains, and likewise draws attention to the referred pains accompanying lesions of the skin, muscles, bones, and joints. Further he carefully describes the motor reflexes which in the form of contractions of the abdominal wall may accompany visceral affections, and likewise the disturbances in the organs which may be set loose by a vegetative reflex action.

However, both Mackenzie's, Head's, and Faber's views of the mechanism of the visceral referred pains and of the nature of the accompanying hyperaesthesias, correspond in all essentials with the theory originally put forward by Lange. And as far as I know, no definitely formulated deviating theory has hitherto been advanced with respect to the mechanism and actual nature of the visceral referred pains, even though much valuable information concerning visceral pains is to be found in many works, amongst others in Foerster's important work on the pathways by which pain is conducted.

When a couple of years ago I began to work out a diagnostic of pain, I soon met with many obscure pain phenomena, presumably of a reflex kind, which could only with difficulty be explained on the current theory concerning the mechanism of referred pain. Hence it seemed necessary to me as far as possible to try to elucidate the facts coming within this field by means of experimental investigations.

These experiments must, according to the nature of the case, be made on human subjects, and since, as already mentioned, irradiated pains and referred pains may just as well

emanate from extravisceral tissues as from the viscera, I have made the experiments, both on patients and on myself, on extravisceral tissue.

By means of a Faradic current numerous experiments were made with stimulation of the skin, and of superficial tendons, muscles and bones of the head, trunk, and extremities. Further, a Faradic current has been passed through the lobe of the ear, the fingers, and the toes. The result was surprising, for in no case was it possible to evoke irradiated pain, but merely pain at the site of stimulation.

To make the matter still clearer I passed a Faradic current through my own teeth, since it is a well-known fact that toothache has a marked tendency to irradiate.

Faradisation with an electrode placed against the edge of the front teeth or the point of the canines produces a pain which strikingly resembles that which is evoked when the dental drill approaches the pulp, but no irradiating pain.

Faradisation of root-treated teeth produces a deep gnawing toothache, but no irradiating pain.

In view of the fact that all degrees, increasing from minimal to maximal strength of current, were employed in the experiments, I think myself justified in contending, on the basis of these experiments, that irradiating pains in human subjects with normal sensibility cannot be produced by faradisation of the skin alone, or of the skin plus the underlying tissues, any more than by faradisation of the teeth.

As every clinician knows, and as has been especially pointed out by Mackenzie, lesions of the tissues in question may, however, be accompanied by pronounced irradiating pains. These pains, then, must emanate from paths belonging to the irritated tissues and these paths must necessarily have become irritated during the faradisation.

The fact that it was, nevertheless, impossible to evoke irradiating pains by faradisation, though all degrees of current were used, would seem to indicate that the Faradic irritation caused an inhibition of the reflex process which must be assumed to underlie the evocation of irradiating pain. A

sensory inhibition has not been produced, however, for even in faradisation by a maximal current only local pains are evoked, and outside the site of irritation even the slightest pin-prick is appreciated with undiminished strength during the faradisation. The inhibition cannot then be of a sensory nature.

But if the inhibition is not of a sensory nature, it must be either of a motor or vegetative kind. Motor reflex inhibition in man may be evoked by faradisation. Thus for instance a human subject exposed to a strong Faradic current is unable to move a finger, even though care has been taken that the current does not touch the finger muscles of the forearm.

But a motor inhibition cannot explain that no irradiating pains are evoked by the irritation experiments, since a motor reflex cannot be assumed to evoke the irradiating pains which are localised to the skin, the bones, and, in toothache, to the other teeth.

By exclusion we then arrive at the result that the above-mentioned inhibition must be of a vegetative kind, which is as much as to say that the reflex process which evokes the irradiating pains must also be of a vegetative kind. The various vegetative inhibitory reflexes are described at length, both in Krogh's Capillary Physiology and in Ipsen's work on arterial reflexes. For the present I must confine myself to referring my hearers to these works with respect to the mechanism of the vegetative inhibitory reflex.

If now, however, it is kept in mind that the visceral pains localised to the abdominal wall are due to a vegetative reflex action, the question arises whether, clinically or experimentally, symptoms may there be observed, which go to show that visceral irritation causes a reflex action of a vegetative nature on the abdominal wall.

In this connection I may remind you of the anemic cutaneous zones, described by me, which accompany visceral lesions. They are co-extensive with the hyperaesthetic zones described by Head and Faber and likewise with the location of the visceral pain, apart from the fact that the widely irradiating visceral pains may extend beyond this zone.

Experimentally I have produced corresponding white zones by visceral irritation of the skin of fishes, and as described in my thesis for the doctorate, I have evoked them not only when the spinal cord of the experimental animal was preserved, but also when the cord had been destroyed before the visceral

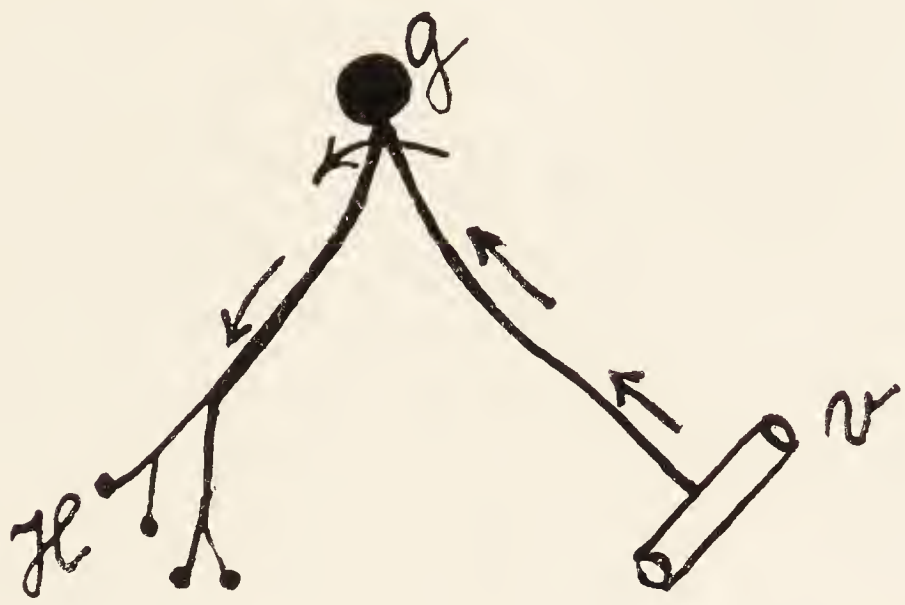

Fig. 4.
Viscero-cutaneous peripheral reflex. — By irritation of a viscus (e. g. part of an intestine, v) impulses are evoked which through long branches of a cell from the sympathetic ganglion (G) are conducted from the viscus to the skin (S) quite outside the central nervous system.

irritation took place, and even after all the cells of the sympathetic ganglia had been paralysed by nicotine painting. As a consequence the reflex must in these cases be supposed to be evoked through peripheral branches issuing from ganglion cells which at the same time give off branches to the viscera and the skin, i. e. it must be a postganglial axon reflex. (Fig. 4). The area of distribution of the branches passing to the skin may in this way be exposed reflexly to vegetative irritation, an irritation which must be supposed capable of producing not only anemia, but also cutaneous hyperaesthesia and pains of a localised kind.

If the cord is preserved, visceral irritation may in fishes cause diffuse blanching of the skin, which must be supposed to be due to a spinal vegetative reflex action. A corresponding

extended paling of the skin may probably also occur in man, but personally I have only observed localised anemic zones which in all probability, like those caused experimentally, are due to a peripheral vegetative reflex action, just as the hyperaesthesia localised to this zone, and the pains localised to the same zone, must be supposed to be evoked by the peripheral reflex in question.

But not all peripheral referred pains can be assumed to be evoked by a peripheral reflex action. As previously mentioned, Foerster has shown that by irritating the peripheral stump of a divided posterior root, pains may be evoked in the entire zone of the root in question, but, be it noted, only in this.

This would seem to show that there are anastomoses between the ramifications of the root in question and those of the neighbouring roots which overlap its zone. But at the same time it would seem to indicate that there is no larger and more peripherally distributed network of nerves. As a consequence it must be regarded as highly improbable that the visceral reflex pains are exclusively due to a peripheral vegetative reflex effect.

It is a well-known fact that the pains which accompany for instance a biliary colic may extend upwards over the chest and radiate right out into the arm, which means that they can extend from the eighth dorsal segment right up to the cervical segments of the arm. A distribution through so many segments cannot, if we take Foerster's experiments into account, be supposed to be due to a reflex action through peripheral ramifications, but must be supposed to be due to a central reflex action — and according to my investigations a central reflex action of a vegetative kind.

On the basis of the experiments and the arguments here advanced it must then be assumed to be predominantly probable that visceral referred pains are due partly to a peripheral and partly to a central vegetative reflex action with a resulting irritation of the tissue to which the pain is localised. —

Important material supporting the correctness of this theory has recently been brought forward through Stürup's

investigations, which were made independently of the aforementioned researches. Stürup chose as his starting-point in a series of investigations on the problems of pain the anemic zones, described by Wernøe, which often accompany visceral pains. He found some difficulty, however, in detecting these faintly pronounced anemic zones, and so he first of all tried to find a method of observation by which the colour contrast would become more conspicuous. He then found that both he and his coworkers, by using Tscherning's glasses, could see the zones distinctly, and his description of these agrees with the description already at hand. But in addition he discovered other less pronounced anemic zones, which I have either overlooked or been unable to detect with the naked eye. These zones were less pronounced or more irregularly delimited. I presume that they are due to a central reflex action, but, as already stated, I have not observed them.

Now in order to ascertain whether the pain which was localised to the most pronounced anemic zone was really evoked by irritation of the tissue in that place Stürup decided to anaesthesise such a zone.

A similar anaesthesia has been produced by a number of other enquirers with the result that the pain ceased; but Stürup produced an anemic zone on himself by introducing a rubber ball into the esophagus and distending it with 40 c. c. of air.

I have personally been present at one of the experiments, and judging by his appearance, this distension caused great discomfort and severe pain. At the same time there appeared on the anterior surface of the thorax a distinct bilateral anemic zone, and the pains were localised to this zone; they were felt to be superficial, localised in the skin and underlying tissue, but anterior to the sternum.

The rubber ball was now removed and this zone was anaesthesised. The ball was again introduced and this time filled with 60 c. c. of air. Now it evidently caused no appreciable discomfort, and Stürup declared that he felt no pain or at any rate very slight pain.

It seems to me that this handsome experiment shows, as conclusively as any clinical experiment can, that the pains caused by the above-described dilatation of the esophagus were due to a reflex process causing an irritation of the peripheral tissue.

Whether this irritation directly affects the peripheral sensory end organs, or whether the vegetative reflex action produces chemical changes which secondarily irritate the tissue with its sensory elements, I do not venture to say.

I consider it improbable, however, that the action should be exclusively chemical, since, for instance, labour pains occur when the uterus is contracted and disappear as soon as the uterus is relaxed, just as the pains in Stürup's experiment set in when the ball was distended and subsided the moment it was emptied of air. — Such a sequence could not presumably be observed if the pains were exclusively dependent on chemical changes produced in the tissue. On the other hand, Lange, Head, Faber, and Mackenzie have already pointed out that the cutaneous hyperaesthesia which often accompanies the pains may persist for a shorter or longer time after cessation of the pain, sometimes for several days, which might perhaps indicate that the tissue had undergone changes of a chemical nature. —

Perhaps both pains and hyperaesthesias are due to a cooperation of different factors. — The question as to the nature of the irritation is still unsolved.

On the other hand, it seems to me that what has been stated above with predominant probability warrants the following conclusions:

Referred pains are evoked by a vegetative reflex action, and the irritation of sensory nerve elements which in the last instance causes the pains occurs in the periphery and not, as previously assumed, in the central nervous system. —

To this must be added, however, that even if the laws governing the rise, localisation, and distribution of the visceral referred pains must thus, in view of what was stated above, be assumed to be of another kind than first supposed by Lange, Mackenzie, Head and Faber, this does not alter the character of the original contribution of these enquirers.

They were the first to observe and investigate the problems of pain in question, and arrive at the solution most probable at that time.

It was especially the works of Mackenzie which aroused my interest in these questions. But in the demonstration of the cutaneous hyperaesthesias, the patient's indication of pain enters as a subjective factor. So I sought to find an objective sign of the presence of visceral pain in the form of a change in the colour of the skin, and since nothing could be observed by direct illumination I used the same method as is adopted in the microscopical examination of uncoloured specimens, i. e. a subdued light. Under this light the white zones corresponding to the extension of the pain, and the distribution of hyperaesthetic zones, if any, could just be detected.

As a matter of course I have especially occupied myself with the anemic zones which must thus be regarded as objective symptoms. They are as a rule triangular and their base reaches the mid-line. If they are due to an unpaired organ fused from two halves (for instance the intestine), they are bilateral and symmetrical, whereas irritation of a lung or a kidney may be accompanied by a unilateral zone.

If, then, one is in doubt as to whether visceral pains are due to an intestinal or a renal disease, the presence of a unilateral cutaneous anemia favours the renal diagnosis, whereas a bilateral cutaneous anemia points to a lesion of an unpaired organ, that is, in this case, the intestine.

Similarly, by observing whether the zones are unilateral or bilateral a differential diagnosis may be obtained for pneumonia-appendicitis, salpingitis-appendicitis etc.

The level of the zones, too, will yield some information. For instance irritation emanating from the stomach will produce

an anemic zone in the epigastrium (cf. fig. 5), while an irritation in the small intestine produces a periumbilical (cf. fig. 6), an irritation of the large intestine an infraumbilical, and rectal irritation a sacral anemic zone. (Fig. 7).

If an epigastric zone in a dyspeptic spreads downwards,

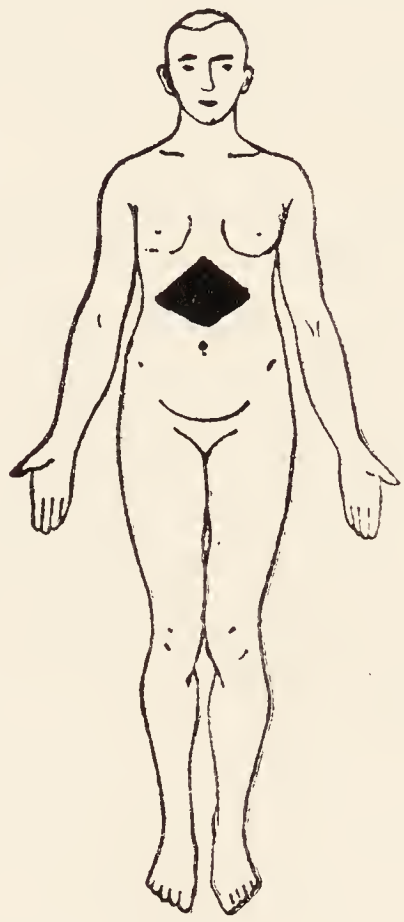

Fig. 5.
Zone of referred pain in gastric lesions.

this is an indication that an irritative process is invading the intestine from the stomach. If the lower limit is fixed, while the anemia spreads upwards, this points towards a stenosis which, proximally to the stenosis, is producing an increasing contraction of smooth musculature. In cardiospasms the lower limit is on a level with the apex of the Proc. ensiformis, in pylorospasm midway between Proc. ensiformis and the umbilicus, in iliocoecal stenosis about midway between the umbilicus and the symphysis, in stenosis of the descending colon just above the symphysis, in rectal stenosis above the os sacrum.

It must be kept in mind that the zones do not change places together with the organs. A patient with gastroptosis has still

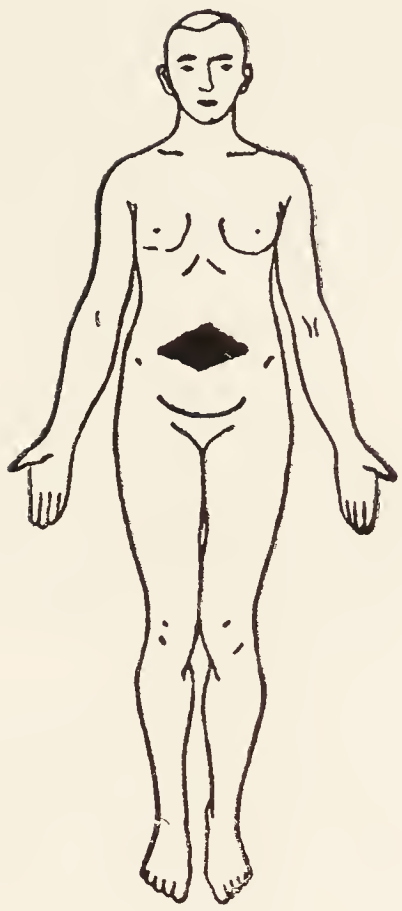

Fig. 6.
Periumbilical zone of referred pains in an affection of the ilium.

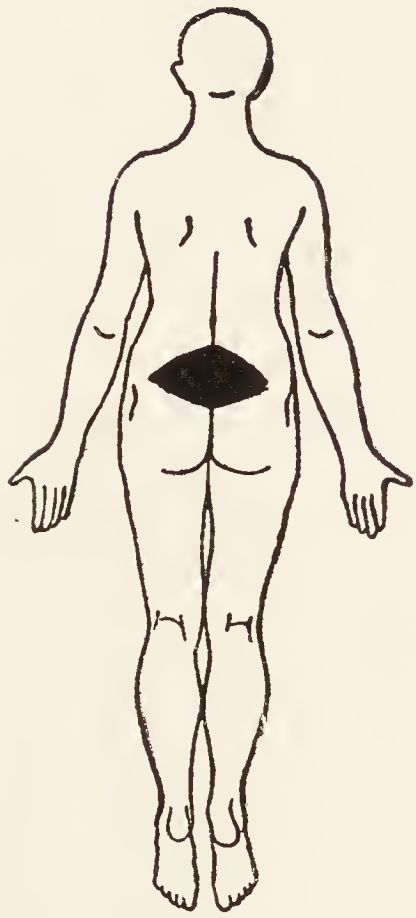

Fig. 7.
Zone of referred pains in a rectal affection.

his gastric zone in the epigastrium, while lesions of high-lying flexures are accompanied by an infraumbilical anaemic zone.

No diagnosis can be based on the anemic zones, but as a diagnostic aid they are, in my experience, of no little value.

More particulars concerning these zones will be given in the special part.

However, to return to the subject of pain, it might perhaps be asked: Of what benefit is it actually to know the detailed mechanism of the visceral pains?

We have already a good knowledge of the type of pain accompanying angina pectoralis, cholelithiasis, nephrolithiasis, and other visceral lesions, and the type will not alter even though the mechanism is analysed ever so thoroughly. —

The reply is that it is nevertheless fruitful to consider the matter from a neurological point of view.

For it is a well-known fact that many visceral affections are accompanied not only by pains in their proper zone, but also by pains extending far beyond this zone. They must therefore be assumed to be due to a central reflex action, evoked through afferent paths ascending through the medulla and on their way in segment after segment connecting up with vegetative centres through reflex paths. This will explain why, for instance, cholelithiasis pains from the hypochondrium spread over the chest and into the arms instead of continuing up the neck (fig. 8). For the second dorsal cutaneous segment is adjacent to the fourth cervical cutaneous segment, while all the intermediate segments go to the arm (fig. 9). The pain cannot bridge this segmental gulf, but radiates into the arm; this means that the afferent paths in question do not pass any large series of segments without giving off reflex paths.

If, therefore, pains in the hypochondrium are accompanied by pains localised to the fourth cervical segments, without pains having occurred in the chest or the arms, there is a predominant probability that this referred pain has travelled

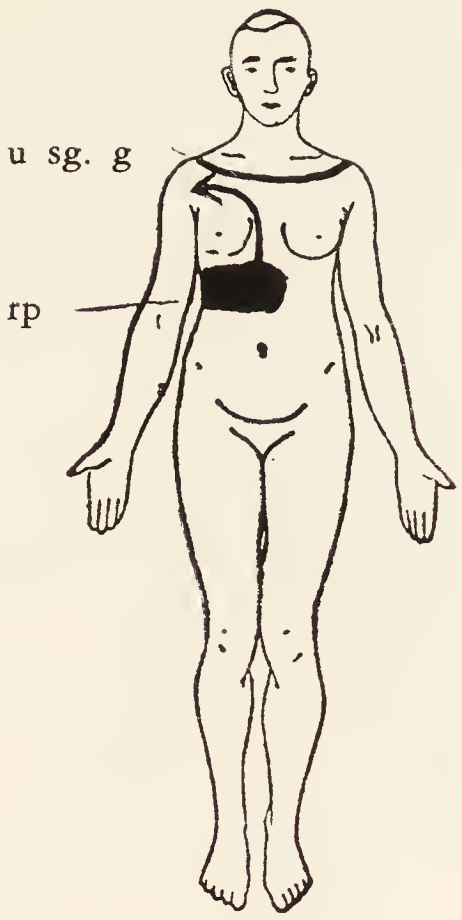

Fig. 8.
Cholelithiasis accompanied by referred pains extending across the chest to the upper cutaneous segmental gap (u sg. g) and thence radiating into the shoulder and arm (rp).

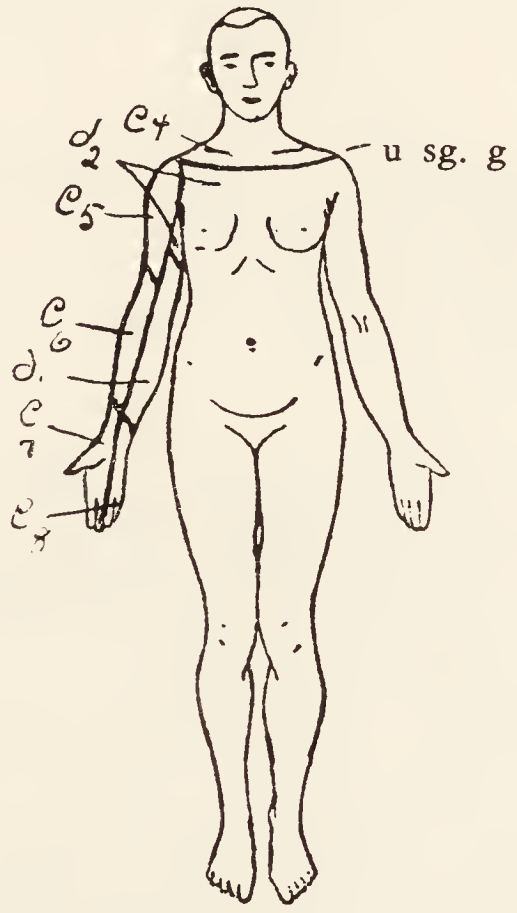

Fig. 9.
IV—VIII cervical and I and II dorsal cutaneous segment. Upper segmental gap (u sg. g).

along phreniscus paths, and the diagnosis diaphragmatic pleuritis is natural. (Fig. 10).

If the patient states that the pains radiate from the hypochondrium up across the chest and further to the neck

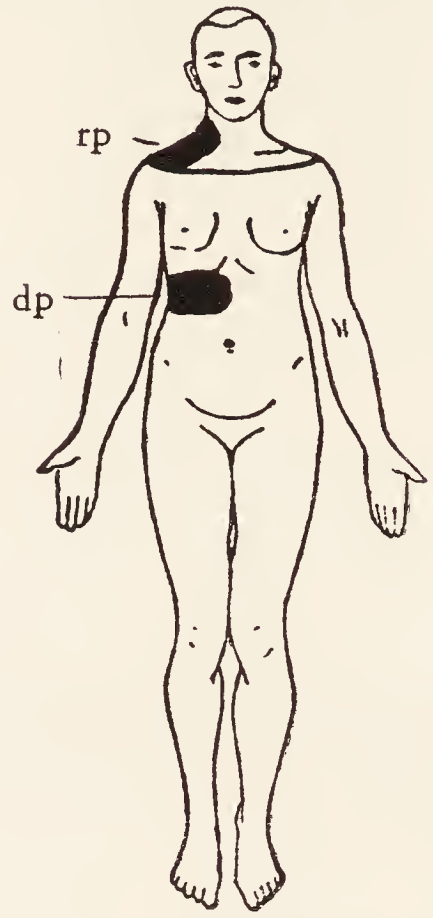

Fig. 10.
Diaphragmatic pleuritis accompanied by direct pain (dp) and referred pain (rp).

without reaching the arms, the pains must be functional. (Fig. 11).

Just as the second dorsal cutaneous segment is adjacent to the fourth dorsal segment, so downwards on the back the twelfth dorsal cutaneous segment is adjacent to the third sacral segment, while the lumbar segments and the first two sacral segments pass to the lower extremities.

As a result centrally caused referred pains cannot spread freely up the back, since in that case they would cross from the third sacral to the twelfth dorsal segment — a segmental gap which is still more difficult to bridge than the dorso-cervical, which is only represented by five segments, whereas the dorso-sacral gap is represented by seven segments (fig. 12).

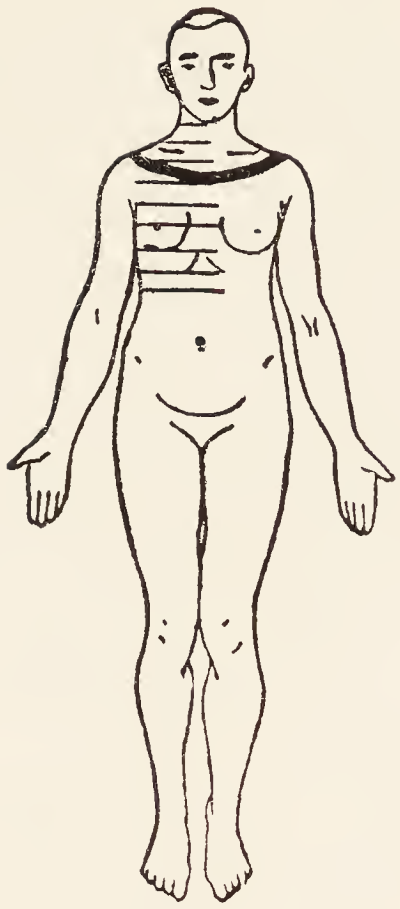

Fig. 11.
Distribution of pain of a functional kind.

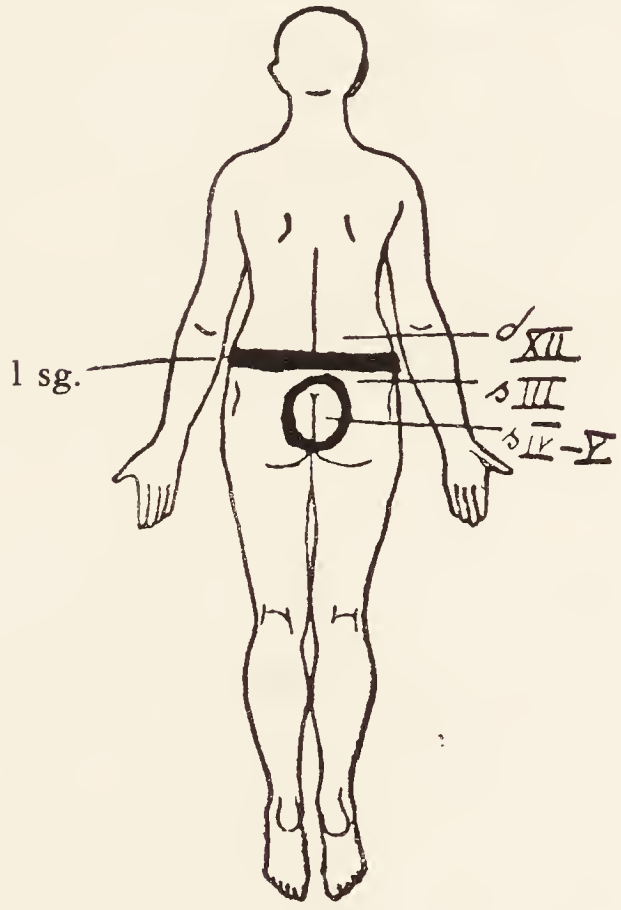

Fig. 12.
At the lower segmental gap (1 sg) the XII dorsal is adjacent to the III sacral cutaneous segment, while all intermediate lumbar and sacral segments pass to the lower extremities.

If, for instance, a lesion of the prostata is accompanied by severe pain, the distribution will in conformity to law be pains over the os sacrum, pains in the lower extremities, and only then ascending pains.

Pains passing from the lumbar region directly to the os sacrum are indicative of the presence of a caudal affection. If pains of that kind accompany a lesion of the prostata, investigation must be directed towards the possible presence of metastases in the column. Similar conditions apply with regard to uterine and rectal pains. But pains which, proceeding from the os sacrum, continue up along the back, must, if local cutaneous muscular and meningeal lesions can be excluded, be regarded as functional.

On the other hand, it is not contrary to law that, for instance, uterine affections are accompanied by isolated pains in the epigastrium or in the corresponding dorsal region, for irritation of the paths which follow Aa. spermaticae int. will probably produce such pains reflexly. It is only the continuous extension of pains from the third sacral cutaneous segment to the dorsal segments which cannot occur reflexly, until pains have radiated into the lower extremities.

In the above-mentioned cases the central reflex action is especially a good guide in forming a diagnosis, in other cases the peripheral reflex action is a better guide. A stone wedged in the ureter may, for instance, evoke referred pains which, because the ureter is innervated by the twelfth dorsal and the first and second lumbar segments, first appear in the twelfth dorsal and then in the first and second lumbar segments, from which the pains can radiate into the lower extremities and thence right down into the sacral segments of the anoperinaeal region.

A distribution of pain of this kind would seem to indicate that the strong irritative effect produced by the stone in the ureter has resulted in an extensive central reflex action. But the distribution of the pain yields no information as to the location of the stone.

If, on the other hand, the patient is suffering from pain

coming on in a series of fits, during which the pains first begin in the twelfth dorsal segment, and then in later attacks begin in the first lumbar and lastly in the second lumbar segment, this suggests that the stone is moving and has finally come into close proximity with the vesica. For according to Head the ureter is innervated by the twelfth dorsal and the first and second lumbar segments, and in accordance herewith a high-lying stone will first evoke pains in the twelfth dorsal cutaneous segment through a peripheral reflex action, a lower-lying one in the first lumbar segment, and a stone approaching the vesica in the second lumbar segment; and this remains unchanged whether or not the pain, through a central reflex action, radiates into the lower segments. Thus information as to the location of the stone may be gained within certain limits by exclusively observing the peripheral reflex action, which usually corresponds to the place where the pain first originates. It is not surprising that this should be so. If it be assumed that a stone or another source of visceral irritation evokes impulses conducted through peripheral ramifications to the cutaneous segment corresponding to the organ, the impulses in question will not be able to pass any further. Hence they are to be regarded as pent-up impulses which, if the influx continues, will be considerably more likely to produce an effect on the limited area of the skin than the centrally conducted impulses, the reflex action of which is distributed over a much larger cutaneous area.

Moreover, it is usually only a smaller part of a segment which is exposed to a peripheral reflex action; but it is also in this limited area that cutaneous anemia can be most plainly observed and hyperaesthesia demonstrated. To this may be added that the zone produced by Stürup in experiments on himself is exactly coextensive in shape and location with the zones described by me, and these again in visceral affections with the most distinctly hyperaesthetic area and with the site of the first pain.

Mackenzie, who had no knowledge of peripheral viscero-cutaneous reflex action, nevertheless used the localisation of

the initial pain as a diagnostic indication, and reports several cases in which this diagnosis of pain stood the test in so far as the stone passed at the time calculated. —

From the example quoted it will, however, presumably appear that while the peripherally caused referred pains are localised pains which do not change their place unless the underlying irritation changes its site, the centrally caused pains are radiating pains which may spread over many segments and radiate into the extremities. The radiation, however, takes place in conformity to law, as described above.

This concludes my discussion of the visceral referred pains, the individual forms of which will be more fully treated in the special part.

Among the extra-visceral referred pains there are forms which are not second in interest to the visceral referred pains.

The reason why I have, nevertheless, preferred to occupy myself especially with the visceral referred pains in this general part, is that the laws governing the localisation and distribution of referred pains seem to me to have been best elucidated for the visceral referred pains.

LECTURE VI.

Pains due to primary motor irritation. Pains due to primary vegetative irritation.

Ladies and Gentlemen.

In view of the fact that muscular pains and muscular hyperalgesia accompanies persistent maximal contractions of voluntary muscles, it is understandable that convulsive states of striated muscles may be accompanied by severe pains and pronounced tenderness of the muscles in question.

Thus the generalised cramps which accompany tetanus often give rise to intense pain, pain which may become intolerable, when there is a paroxysmal aggravation of the contractions owing to the sudden influence of light or sound, a fit of coughing, a swallowing movement, or merely if the patient is exposed to a sudden contact or other sudden external influence.

Similarly the carpopedal spasms which accompany tetanus as a rule give rise to pain, and those of you who, after long dancing, have been attacked by tonic cramps in the muscles of the calves, will no doubt have retained a vivid impression of the intense and unpleasant pains that may be caused by muscle spasms. The primary irritation in these cases is from motor nerve elements, which then secondarily give rise to muscular pains causing spasms.

The pain is localised, though somewhat diffusely, to the region of the muscle and thus at some distance from the primary site of irritation.

Consequently the pains in question must be classed as indirect pains; but since they can neither be regarded as

projected pains nor referred pains, it seems to me natural to place them in a special class.

As regards the pain mechanism itself, there can hardly be any doubt that both mechanical and chemical factors assert themselves here. As has been emphasised especially by Helweg, it must be supposed that forced persistent muscle contractions may cause an accumulation of toxic substances, which influence the muscular sensory nerve elements and so give rise to pains. The so-called myopathiae e labore, which may persist for several weeks or months, must be supposed to be caused in this way. In the special part Helweg's theories will be mentioned at greater length in the section on sciatica.

The pains arising from muscular spasms can, however, hardly be caused exclusively by chemical agents; mechanical factors, too, must be assumed to assert themselves, for pains evoked experimentally by persistent maximal muscle contraction disappear the moment the muscle is relaxed.

Such a rapid subsidence of the symptoms would not be conceivable if the pains were due solely to a chemical influence of the muscular sensory nerve elements.

In this group of pains are only classed the muscular pains caused by primary motor agencies, while muscular pains due to primary infectious or toxic influence on the muscular sensory nerve elements do not belong to this class, any more than the muscular pains arising from reflex action.

In the special part the cases coming under this head will be discussed more fully. Here I shall merely point out that the pain in the former cases arises after preceding forced muscular contractions, whereas pains may occur as the first symptom both in myosites and in neuritic affections.

Although pains occurring secondarily after primary motor irritation may present difficult problems, conditions are even more complicated when the pains emanate from the vegetative nervous system, which in all probability represents a much greater pain factor than has been generally supposed. —

In the first place all pains due to psychic agents are started through the vegetative nervous system. It is a well-known fact

that emotion may greatly influence the vegetative nervous system with resulting symptoms of a vasomotor and muscular kind. We blush with pleasure and turn pale with fear. The heart throbs with anticipation, nervousness before exams may be accompanied by diarrhea.

In neuropathics psycho-vegetative symptoms of this kind frequently occur in an intensified form and accompanied by pain.

In this respect I need only remind you of psychically caused cardiospasm, pylorospasm, and all the other spasms which may affect the different parts of the digestive tract. Further, every clinician knows that a psychic influence through the vegetative nervous system may evoke painful uterine contractions, which may sometimes result in abortion, as has been observed in besieged towns.

Painful vesicular spasms, too, may be evoked through psychic agents. Thus it is often stated by patients with morbus Raynaud that the very painful fits of vasoconstriction which accompany this disorder, arise after agitation.

But central vegetative centres can also be influenced in other than psychic ways.

Thus generalised anemic conditions often evoke vaso-constriction, which in all probability is due to an irritative state of central nervous vegetative centres.

In fishes, at any rate, I have observed that bleeding is accompanied by universal cutaneous vasoconstriction and also by pigment contraction if the bleeding occurs when the brain and cord are preserved. If, on the other hand, the brain and cord are destroyed before bleeding is induced, this as a rule produces hardly any cutaneous vasoconstriction, despite the fact that the sympathetic ganglia and the peripheral sympathetic paths are preserved.

This shows very plainly that the central vegetative nerve elements in fishes are more sensitive to anemia caused by bleeding than the peripheral vegetative nerve elements and the vascular muscles themselves.

These experiments prove nothing with respect to the

corresponding conditions in man, but nevertheless they give us a hint as to where in the vegetative system the irritation is to be sought which in anemic conditions produces cutaneous vasoconstriction.

In so far as the said vasoconstriction is accompanied by pains, these pains are thus probably to be regarded as neuro-vegetatively caused pains.

It has previously been mentioned that gastric crises and other organic crises must be assumed to be due to discharges from central vegetative centres which thus give rise to the visceral spasms that are the actual cause of the pains. The tabetic crises have already been mentioned, and I shall not here go more fully into the questions relating to them.

Staemler in several cases of morbus Raynaud found pronounced degenerative changes in the sympathetic ganglia. Thus it seems that painful contractions of the vessels of the fingers may emanate from them, whereas, for instance, the symmetrical vasoconstriction which sometimes occurs in syringomyelia is not usually accompanied by pain. The explanation is presumably as follows.

If an irritative affection of sympathetic ganglia causes convulsive contractions of the vessels of the fingers, these contractions will presumably give rise to an irritation of sensory peripheral end organs, from which impulses of pain can be conducted unimpeded through intact sensory paths to the cerebrum.

In patients suffering from syringomyelia, vascular spasms may also be freely evoked by an irritation emanating from central vegetative ganglion cells, which through the anterior root, rami communicantes albi, the sympathetic ganglia and the peripheral vegetative paths reaches the vascular musculature of the fingers; but the pain impulses normally following upon vasoconstriction will not as a rule in this lesion be conducted through the central nervous system, because of the onset of a degeneration of the posterior horn. Thus a central organic lesion of the nerves may give rise to painless vascular cramps, while vascular cramps due to a lesion of the central chain of

ganglia will be accompanied by pains, because the sensory paths running along the nerve trunk are uninjured. This circumstance has long been turned to good account in clinical medicine in cases where it was doubtful whether a cutaneous vasoconstriction was due to syringomyelia or morbus Raynaud. For painless vasoconstriction points towards a syringomyelia, while vasoconstriction accompanied by severe pain is indicative of morbus Raynaud.

In view of the fact that an irritative affection of the central chain of ganglia seems able to give rise to a painful cutaneous vasoconstriction, it might be expected that visceral spasms might arise as a result of such an affection. There is hardly any doubt, either, that painful paroxysmal visceral spasms may be evoked by an affection of the central chain of ganglia.

Thus painful visceral spasms, and even ileus-like cases, often accompany prevertebral tumors and prevertebral imflammatory lesions. In these cases it is, however, difficult to decide whether the visceral symptoms are due to an irritation of the central chain of ganglia or of the visceral nerve trunks. However, in so far as prevertebral lesions are exclusively accompanied by visceral symptoms, it is most likely that they are due to an irritation of the visceral nerve trunks. If pronounced root pains accompany the visceral symptoms, it is natural to regard these symptoms as the outcome of an irritation of the nerve roots, but if the visceral pains are accompanied by cutaneous vasoconstriction without accompanying root symptoms, then an irritation of the central chain of ganglia is most likely to be the cause, since such an irritation both affects the vegetative paths running to the viscera and to the skin, or rather those ganglion cells which form the starting point for the aforementioned peripheral visceral and cutaneous paths. However, diffuse lesion, for instance an infiltrating tumor or a diffuse phlegmonous process, may perhaps at the same time attack both the nerve roots, the central chain of ganglia, and the visceral nerve trunks, and in accordance herewith there will be a possibility that new

combinations of symptoms may appear in the direction of simultaneous root symptoms, visceral spasms, and cutaneous vasoconstriction.

If pathological anatomists would give attention to these conditions and subject the nerve roots, the central chain of ganglia, and the visceral nerve trunks to a careful examination at autopsies whenever there is a history of colics of obscure origin, the conditions in these cases might possibly be elucidated.

While the pains which may emanate from the central chain of ganglia and from the visceral nerve trunks greatly need investigation, conditions are somewhat clearer in respect of the pains which arise secondarily after a primary irritation of the vegetative paths following the long cerebrospinal nerve trunks.

If one of the long nerve trunks in man is irritated by means of a Faradic current, no pronounced vegetative symptoms will as a rule appear. In all probability the reason is that both vasoconstrictive and vasodilating effects are evoked by the irritation and hold each other in check.

For electrical stimulation affects both the vegetative and the sensory paths. An irritation of the vegetative paths will give rise amongst other things to vasoconstriction, while irritation of the sensory paths may by an antidromic effect cause vasodilatation. Simultaneous irritation of the vegetative and sensory paths thus involves effects of diametrically opposed kinds, and in so far as the two effects are of equal strength, they will cancel each other. No visible effect will then appear, apart from the accompanying motor effects, which are disregarded in this connection.

Such a balance between the vasodilator and the vasoconstrictive effects in the irritation of spinal nerves does not, however, occur everywhere in the animal kingdom. Thus, on the skin of fishes, irritation of a spinal nerve by a Faradic current will produce a pronounced vasoconstriction and a pigment reaction, but no visible dilatation. Here, then, we do not find the aforementioned balance between the contracting and dilating forces, probably because the sensory system in

these animals is but slightly developed compared with the vegetative nervous system.

But in man, at any rate, I have not succeeded in producing any distinct effect in the direction of cutaneous vasoconstriction or vasodilatation by faradisation through the skin of long nerve trunks. On the other hand, it is often observed that partial damage to a peripheral nerve is accompanied by pronounced vasomotor disturbances, and especially when the injury is of the median nerve.

As far back as in 1864 Weir Mitchell described lesions of this kind under the name of causalgia, cases which he had observed in the American Civil War. The term causalgia is perhaps not very happy. It easily suggests the Latin word causa, meaning cause. Causalgia is, however, derived from two Greek words; kausis, which means burning, and algos which means pain, and this designation is in so far happy as burning pains of great intensity often accompany the partial nerve lesions. Lesions of this kind were observed in great numbers during the world war, and in time of peace, too, cases of this kind frequently occur.

There are two forms of causalgia, a vasoconstrictive and a vasodilator form. The former, as indicated by the name, is accompanied by cutaneous vasoconstriction. The skin is cold owing to spasms of the arterioles and consequent diminished blood supply. It is likewise cyanotic because of stasis in the capillaries and small veins. The secretion of sweat is reduced and the skin is dry with fissures and hyperkeratosis.

The other form is accompanied by vasodilation. The skin is red and perspires freely, it is hot and frequently thin and glossy. While the vasoconstrictive form is often accompanied by more or less pronounced sensory and motor deficiency symptoms, the vasodilator form is unaccompanied by such.

In both forms the pains may occur by fits and increase to a great intensity. In the vasoconstrictive form the pains are generally alleviated by the local application of warmth, while the influence of cold, for instance in the form of cold compresses, generally reduces the pain in the vasodilator form. Finally

it may be added that the periarterial sympathectomy introduced by Leriche often ameliorates the condition in the vasoconstrictive forms, whereas a similar improvement is not observed in the vasodilator forms.

If now, on the basis of the above, we were to attempt to analyse the mechanisms of pain which must be supposed to be the rule in the two forms, it is immediately understandable in respect of the vasoconstrictive form that vascular spasms may evoke severe pains which may be alleviated by means of a peripheral vasodilator influence. But it can hardly be supposed, as Foerster would have it, that the vasoconstriction is due to the loss of vegetative innervation; for polimyelitis may be accompanied by vascular spasms, too, with a resulting cold and cyanotic skin, though attacks of pain are not therefore among the usual symptoms of polimyelitis. Whether now we assume, with Joh. Ipsen, that the cutaneous vascular spasms accompanying polimyelitis are due to a loss of spinal vegetative inhibitory mechanisms, or we regard the cutaneous vasoconstriction accompanying polimyelitis as a result of the independent action of peripheral mechanisms, we cannot disregard the fact here exemplified that the loss of vegetative innervation is accompanied by vasoconstriction which is not painful, though sensibility in polimyelitis is well preserved. Incidentally, a similar painless cutaneous vasoconstriction may also often occur in monoplegias and hemiplegias of cerebral origin, which suggests that the vasoconstriction accompanied by severe pain is not to be regarded as a deficiency symptom but as an irritation phenomenon, and this probably is also the case with the causalgic vasoconstriction. But the pain is hardly due to vasoconstriction alone. That anemia of the tissues which accompanies vasoconstriction is probably in part responsible for the pains, since it is a well known fact that the sudden invasion of an anemia, caused for instance by an embolus in the artery of an extremity, may cause intense pain in the tissue supplied by the artery in question.

Finally, as a third pain factor must be added the direct irritation of the nerve trunk produced by the lesion. In all

probability, however, the chief pain factor is the vasoconstriction evoked through the vegetative irritation; for the pain is alleviated in a striking degree, if the extremity in question is exposed to a vasodilating peripheral effect.

If we turn to the vasodilator form of causalgia, the pains in this form must also be supposed to be peripherally caused, since a peripheral application of cold considerably alleviates and diminishes them. In all probability the reduction of the pains is due to the circumstance that the application of cold, by producing vasoconstriction, counteracts the hyperaemia which is present. Thus the hyperaemia must be supposed to represent a pain factor of importance, but not in the same way as the aforementioned vasoconstriction in the form of causalgia. For while vascular spasms give rise to pain, vasodilatation is painless. Not only the psychically caused redness and the cutaneous hyperaemia produced by the application of warmth, but even the cutaneous hyperaemias accompanying painful cutaneous irritation are painless. One may convince oneself of this by observing the dermographic reactions.

As shown by Krogh, scratching with a needle produces a cutaneous hyperaemia in the shape of a stripe (dermographia rubra) (fig. 13) through a peripheral sensory reflex action, and a stronger irritation will cause a more diffusely extended hyperaemia (dermographia reflexiva) which, according to investigations by Krogh and Rehberg, is also in all essentials due to a peripheral reflex action through long much ramified sensory paths (fig. 14), even though a feebly pronounced central vasomotor inhibitory reflex would seem to contribute its share.

Dermographia rubra, and especially dermographia reflexiva or reflex erythema, are well suited for the study of the relation between vasodilatation and pain: for if the scratches are made quickly, the pain will subside before the erythema appears, and it will then be possible partly by experiments on oneself, and partly by questioning the patient, to ascertain whether the vasodilatation caused by painful stimulation is accompanied by pain.

Fig. 13.
Dermographia rubra: Scratching with a needle produces cutaneous vasodilatation in stripes, which according to investigations by Krogh is due to a peripheral reflex through short sensory paths. A more vigorous irritation may produce diffuse cutaneous hyperaemia, the so-called reflex erythema.

In my investigations, which comprise about 2000 cases, vasodilatation was in no case accompanied by pain; hence vasodilatation must be regarded as a painless phenomenon even if it is produced reflexly by irritation of sensory paths.

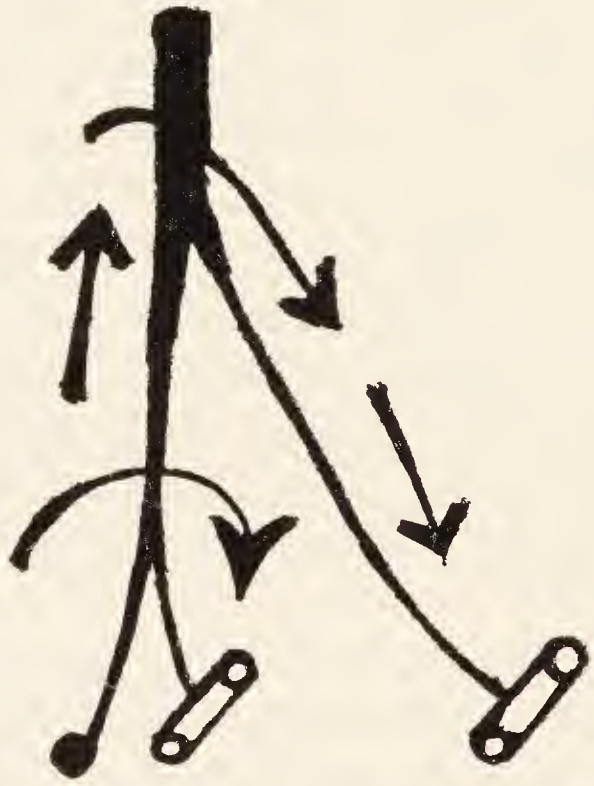

Fig. 14.
Reflex erythema, according to investigations by Krogh and Rehberg, is in all essentials due to a peripheral reflex through long sensory paths, during which a reflex action through short paths must, however, also be supposed to take place.

In the vasodilator form of causalgia the vasodilatation itself cannot therefore be supposed to give rise to pain, whereas the vasodilatation and the accompanying arterial hyperaemia may cause an increased irritability of the tissues. One may convince oneself of this merely by holding a finger in warm water until it becomes red and hot.

If the skin is then tested by means of pin-pricks, an increased irritability will be found, which is coextensive with the hyperaemic area.

The hyperaemia accompanying the vasodilator form of causalgia will thus probably cause a change in the pain receptors, so that an irritation of these may give rise to intensified pain, but the irritation of the pain receptors must be due to a factor other than the hyperaemia.

It seems to me most probable that the irritation of the pain receptors originates from the primary lesion of the nerve trunk, and that antidromic impulses evoke this irritation, since impulses conducted backwards, according to Foerster's aforementioned experiments, are capable of irritating the pain receptors and thus of producing pain. But at the same time they also, as shown experimentally by Bailis, Doi, Krogh, Harrop, and Rehberg, cause a vasodilatation with a consequent hyperaemia.

Antidromic impulses must thus be supposed to be capable of producing both irritation of the pain receptors with consequent pain, and, further, the hyperaemia which intensifies the pain (fig. 16). On this assumption it is understandable that a vasoconstrictive effect may alleviate the pains by counteracting the hyperirritability of the pain receptors, which is a result of the arterial hyperaemia.

As already mentioned, causalgia occurs especially in lesions of the medianus, but it may also accompany other partial trunk lesions.

To this must be added, however, that other trunk lesions also may be accompanied by vegetative symptoms though usually in a less pronounced form. But both vasoconstrictive and vasodilator phenomena very often accompany both infectious

and tonic forms of neuritis, so it must be remembered that trunk pains are not only due to irritation of the nerve sheath and nerve paths, but the pains caused by vasomotor disturbances, as described above, are an additional cause.

Consequently, not only in some few rare cases but probably

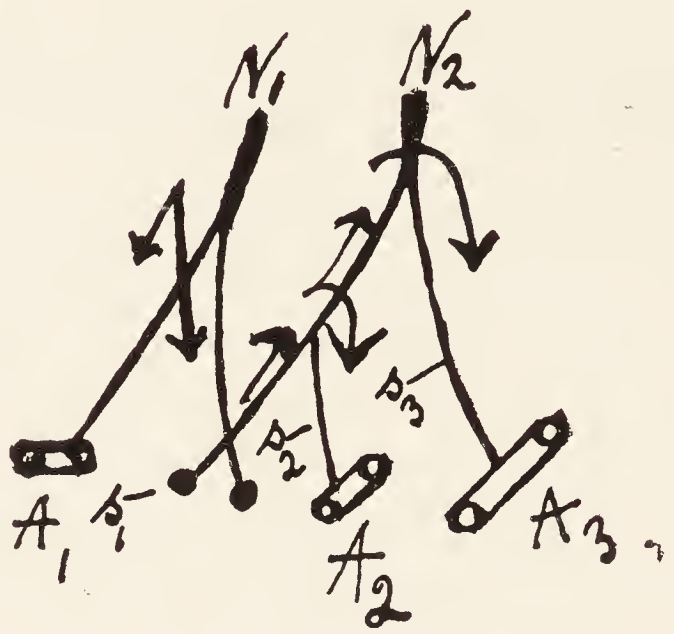

Fig. 15.
Hypothesis to explain the mechanism of pain in the vasodilator form of causalgia.
Irritation of the sensory paths following nerve trunk N_1 evokes antidromic impulses which partly by anastomoses irritate the terminal ramifications of N_2 and partly produce hyperaemia by affecting the cutaneous vessels, A_1, a hyperaemia which causes a change of tone in the pain receptors and hence an increase of the pains evoked by N_2. Probably the peripheral hyperaemia is increased by a peripheral reflex action through the peripheral reflex mechanisms demonstrated by Krogh and Rehberg (s_1, s_2, A_2; s_1, s_3, A_3).

in many more cases of peripheral nerve lesions than is generally supposed, the vegetative nervous system is a pain factor of importance.

From the above it will presumably already have appeared that a primary irritation of the vegetative nervous system may in many cases be the cause of fits of pain of the most troublesome kind. In the special part of this work we shall mention the reasons speaking for the assumption that both the pains

of migraine and of the malignant trigeminus neuralgia are due to a primary vegetative irritation, and if we add to this that all referred pains, as previously mentioned, must be supposed to be evoked through a vegetative reflex action, it will be evident that the vegetative nervous system constitutes a very important factor in mechanisms which may produce fits of pain of the severest kind.

The question of the significance of the vegetative nervous system as a factor productive of pain is of interest not only in a theoretical but also in a practical respect.

With the immense development which has taken place in neuro-surgery during the last decades, the vegetative nervous system has also in great part been drawn into this field, and operative incisions are made both on the peripheral vegetative nerve paths and on the central chain of ganglia.

It is then in view of the indication for operation important to be able to distinguish the vegetative sensory forms of pain from the forms belonging exclusively to the sensory nervous system.

LECTURE VII.

Psychogenic pains. — Combined pains. — Concluding remarks.

Ladies and Gentlemen.

By psychogenic pains are meant, in the following, not only such as arise from psychic traumata, but also, in general, pains having a psychic cause. —

Primary psychic influences may give rise partly to motor, partly to vegetative irritation phenomena and thence to pain.

Thus after psychic shocks painful contractions are often observed and the painful muscle infiltrations so frequently occurring in neurasthenics are likewise probably due to circumscribed muscle contractions of psychic causation.

At any rate a colleague of mine told me that at one time he was treated with massage for painful and tender muscle infiltrations which the masseuse could without difficulty demonstrate by palpation.

One day, however, he fell asleep in the middle of the treatment, and at the same time all the infiltrations disappeared. This presumably means that they were due to contractions and not to edematous processes, since such could not be supposed to undergo any change upon the incidence of sleep. If we add to this, that the infiltrations are known to be fugacious, appearing now in one region, now in another, it will presumably be reasonable to regard the infiltrations as well as the accompanying pains as due to psychic causes.

As previously mentioned, psychic influences may, however, give rise to vascular and visceral spasms through the vegetative nervous system, and thus again to pains, sometimes even so severe that they have been regarded as signs of the presence

of serious organic lesions, the patients in question having been subjected to operations.

However, for psychic influences to give rise to vascular or visceral spasms of the above-mentioned painful kind, there must in the patient in question be a labile nervous system, for normally neither psychically caused vascular contractions nor visceral contractions are accompanied by pains which can be mistaken for the various visceral colics.

In neuropathics, however, psychic influences will for several reasons produce heightened reactions. In the first place, as pointed out by Oppenheim, psychic influences may in neuropathics produce abnormally strong psychophysical responses of a motor or vegetative kind. —

In the second place, the visceral spasms evoked in this way may for instance give rise to pains which are felt with abnormal intensity owing to the presence of a psychic hyperaesthesia, and finally and thirdly, these pains may cause abnormally strong manifestations of pain in the shape of loud moaning or other strongly marked intimations of pain.

If it is desired to form an estimate as to which factor is of the greatest importance when intolerable psychogenic visceral pains are manifested, the main stress must be laid on the observation of the phenomena known by experience only to accompany organically caused visceral pains when these occur with great intensity. To these phenomena belong in the first place the centrally caused contractions of the muscles of the abdominal wall, the so-called viscero-motor reflexes which accompany not only peritoneal affections but also severe attacks of biliary colic, renal colic, pylorospasm and other painful visceral spasms.

The abdomen may then become as hard as wood, a phenomenon very rarely observed in painful paroxysms of psychic origin, a fact which would seem to indicate that the irritative impulses conducted to the central nervous system during attacks of pain due to organic causes are much stronger than those which generally occur in fits of psychogenic pains. Other facts point in the same direction. Thus the centrally

conditioned referred pains irradiating to the extremities frequently occur in attacks of visceral pain due to organic causes, but rarely in attacks of pain of psychic origin. In addition, respiration is usually affected in pain due to organic causes, so that the patient is unable to breathe and talk freely. Frequently, when the pains approach their maximum, talk is interrupted in the middle of a sentence owing to inhibition of respiration, an inhibition which is followed by intensified breathing and sometimes by loud moaning. But at the height of the attack the patient is unable to speak freely and naturally owing to the altered respiration. Conversely, patients suffering from psychogenic pains are usually able to describe their pains loudly in appropriate words and unbroken sentences. All this suggests that the irritation which flows to the brain during attacks of psychogenic visceral pains is not usually so intense as that which is due to pronounced organically conditioned attacks of visceral pain.

Since, nevertheless, psychogenic visceral pains are frequently stated even by intelligent patients to be extremely pronounced, the strength of the pain must be supposed to be mainly due to the presence of a hyperaesthesia enabling irritative impulses, which would normally only cause moderate pain, to give rise to pains of great intensity.

The presence of a psychic hyperaesthesia will also explain the fact that severe and persistent pain often arises in neuropathics from small and relatively unimportant organic affections. Scars which have healed ideally, well-treated sinuites, distorsions, fractures, and minor lesions, apparently healed in the normal way, in neuropathics, give rise to protracted and troublesome pains, probably due to a minimal irritation from the site of the lesion, which owing to the presence of a psychic hyperaesthesia causes troublesome pains.

Similarly, previous affections of the stomach and intestine, of the biliary ducts and of the urinary and genital organs may have left behind small changes which in the presence of a psychic hyperaesthesia may give rise to frequent or continuous pains.

Particularly when people who have sustained injuries in accidents, and claim damages, complain for months or years of pains owing to previous trifling lesions, it is natural to explain these as the outcome of an accentuated psychic hyperaesthesia caused by the thought of compensation.

However, spontaneous pains, too, will frequently arise in patients with psychic hyperaesthesia. They may occur in any part of the body, now as superficial, now as deep-seated pains, often as headache, and often as backache, but also frequently in the extremities, in the chest or the abdomen. Sometimes there is a distinct hyperaesthesia at the site of the pain. Thus a limited hyperaesthesia often occurs in the crown or some other part of the head, just as hyperaesthesia plantaris is frequently present; but in other parts of the body, too, circumscribed hyperaesthesias partly of the skin, partly of deeper-lying tissues are often demonstrable.

Spontaneous pains of psychic origin may, however, also occur unaccompanied by hyperaesthesia. In that case they are termed pain hallucinations or psychalgias. This means that there is a direct irritation of pain-percipient centres. (Perhaps, however, there is in these cases an abnormally heightened perception of the impulses normally issuing from the tissue rather than an irritation of pain-percipient ganglion cells).

If now we have to consider how psychogenic pains in general can be distinguished from pains organically caused, first of all a thorough objective examination is needed, in order to ascertain whether any local organic changes will furnish a natural explanation of the presence of pain.

If none such are found, a general examination must be added so as to demonstrate, if possible, any changes which, through a reflex action or projection, might be the cause of the pains in question.

The neurological investigation must in these cases not only be directed towards deficiency symptoms; but pains and hyperaesthesias, too, must be neurologically tested. For even if pain is the only symptom present, it must be supposed that, if it is precisely localised to the area of a nerve trunk, it is due to

an irritative trunk affection, just as it must be regarded as predominantly probable that an irritative root affection underlies the pains which show a radicular unilateral or bilateral distribution. Further, unilateral facial pains combined with pains localised to the opposite half of the body must be supposed to be due to an irritative pontine affection.

Other forms of crossed sensory irritation symptoms, too, suggest the presence of an underlying organic lesion.

Unilateral pains in the head combined with paraesthesias in the opposite half of the body may thus be the only symptom accompanying a unilateral cortical affection, just as unilateral root pains combined with pains in the opposite half of the body may be the first symptom of a hemilateral lesion of the cord.

Conclusions concerning the underlying lesion may also be based on the way in which the pain spreads. Visceral reflex pains which, on spreading from the dorsal segments, respect the dorso-cervical and the dorso-sacral segmental gap will thus as a rule be organically caused, especially if the pains are accompanied by pronounced viscero-motor reflexes and by inhibited respiration and difficulty in talking, even though no other symptoms of an organic lesion can be demonstrated.

Distribution of another kind may also furnish important diagnostic holds; pains in the arms or legs which spread from one trunk area to the neighbouring area without going beyond this, suggest the presence of a plexus lesion, whereas extension to the symmetrical trunk area points in the direction of a polyneuritis affection.

Pains which in the course of a lengthy period spread successively but connectedly over a whole series of segments must be assumed to be due to a diffuse central organic lesion, a meningitic or a myelitic process; and pains localised to a single root zone which spread from there to the symmetrical root area suggest the presence of an extramedullary tumor or other local process spreading transversally in the spinal cord.

But if no sign of organic lesions can be demonstrated, and if the pain does not show the localisation and distribution

characteristic of well-known types of pain caused by organic lesions, it must be considered likely that the pain is of psychic origin. This assumption receives support if the pains prove to have arisen in connection with a psychic trauma, or if they are dependent on the mood of the patient, so that they only occur when he is tired, depressed, and irritable, whereas they disappear in his brighter periods. Signs of a considerable psychic hyperaesthesia otherwise demonstrable in the patient will also support this diagnosis.

Other symptoms point in the same direction.

If the pains, as not rarely happens, are stated by healthy-looking patients always to have been present, or if pains are stated to occur all over the body, there can hardly be any doubt that they must be regarded as psychogenic. It is also predominantly probable that pains stated to come from the viscera but, contrary to law, spreading from the dorsal segments directly to the neck without first extending to the arms, belong to the psychically conditioned pains.

A conspicuous disproportion between the pain responses of the patient and the asserted strength of the pain points in the same direction. This can be observed when smiling patients apparently enjoying perfect health state that they are subject to intolerable pains, and, conversely, when the patient's reaction to trifling affections, which have been shown by experience to run an almost painless course, is so violent that the visible manifestation of pain is out of all reasonable proportion to its cause.

Thus it is not rarely the case that patients kick and fight as soon as the physician tries gently to test their sensibility by pin-pricks or merely by passing a brush over the skin. Reactions of this kind do not accompany even the most pronounced somatic hyperaesthesia or hyperalgesia. The patient may start upon painful pressure or pricking, but persistent kicking or fighting does not occur in individuals with healthy nerves on account of a pin-prick or other brief painful stimulation.

It is always of importance to ascertain whether functional pareses or functional anaesthesias are present, whether the

pharynx reflex or the naso-facial grimace reflex has been lost, whether astasia-abasia, psychic disorders, or other symptoms can be demonstrated as signs of the presence of a functional nervous disorder.

It is true that even so the pains of which the patient complains may still be due to organic causes; but if no sign can be shown of an organic lesion, it must nevertheless be considered likely that the pains in question are to be regarded as the outcome of the functional nervous disorder shown to be present, and less likely that they should be due to an organic lesion which does not present any other symptoms.

It is one of the most difficult tasks of the clinician to distinguish with certainty between pains due to psychic and pains due to somatic causes. A careful study of the history is needed, besides a thorough objective examination and a knowledge of the rules and laws according to which organically caused pains are localised and distributed.

Finally I shall briefly discuss the third and last of my three main groups of pains, the combined pains.

It has previously been mentioned that pains may occur as combined direct and indirect pains. Thus the pains accompanying neuritis consist partly of direct pains emanating from the nerve sheath, partly of projected pains emanating from the long nerve paths. (Cp. figs. 16 and 17).

Local meningeal lesions may give rise to direct pains, as is often found in spondylitis, owing to the irritation of the peripheral sensory end organs innervating the meninges, and to projected pains arising from pressure on the nerve roots or on the medulla. Extramedullary and extracerebral tumors, too, may cause direct pain owing to pressure on the meninges, and projected pain arising from pressure on the medulla or on the nerve roots.

Further, direct pains may be combined with referred pains. Thus affections of the teeth are often accompanied both by

local and by irradiating pains, which may spread partly to other teeth partly across the cheek as far as the temple. Similarly, furuncles may give rise to direct pains combined with referred pains which may spread widely. Other inflammation-like lesions of tissues cerebrospinally innervated

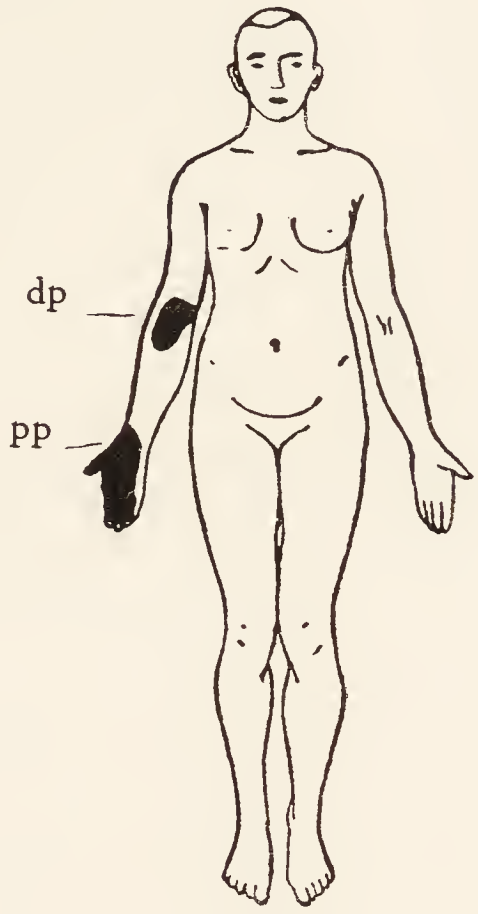

Fig. 16.
Medianus affection accompanied by direct (dp) and projected pains (pp).

are very frequently accompanied by combined pains, and if visceral lesions are accompanied by peritonitis, the peritonitis will give rise to direct pains while referred pains may at the same time emanate from the viscera.

Often direct pain is accompanied by local tenderness. In such cases the point of origin of the pain can fairly easily be found. In other instances no local tenderness is present, as for example in intracranial and intraspinal lesions. In these cases the local diagnosis, if pain is the only symptom present, must be based solely on the patients' indications of pain.

It must be remembered that direct and indirect pains may

occur simultaneously in the same part of the body without on that account having any connection with each other. These multiple pains will, however, be left out of consideration in this connection, for the group of pains here posited comprises only cases of organic lesions which at the same time give rise

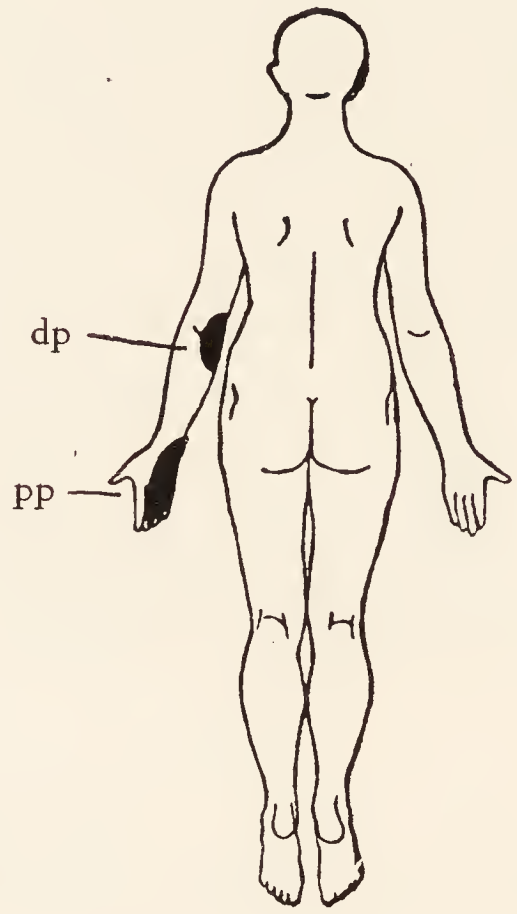

Fig. 17.
Ulnaris affection accompanied by direct pain (dp) and by projected pains (pp).

to direct and to indirect pains, as is the case with the examples given above.

In the first lecture it was mentioned that as regards level and transverse extension the patient is able to localise direct pains with a degree of certainty sufficient for diagnostic purposes, while the localisation as to depth is very doubtful. As a consequence the diagnosis of isolated, deeper-lying direct pains will always be marked by uncertainty.

Nevertheless they may, when they occur in conjunction with indirect pain, often render possible a probable focal

diagnosis which cannot be based on isolated indirect pains alone.

Indirect pains localised to the area of distribution of a trunk, must thus, as already mentioned, be assumed to be due to an irritative trunk affection, but they furnish no information

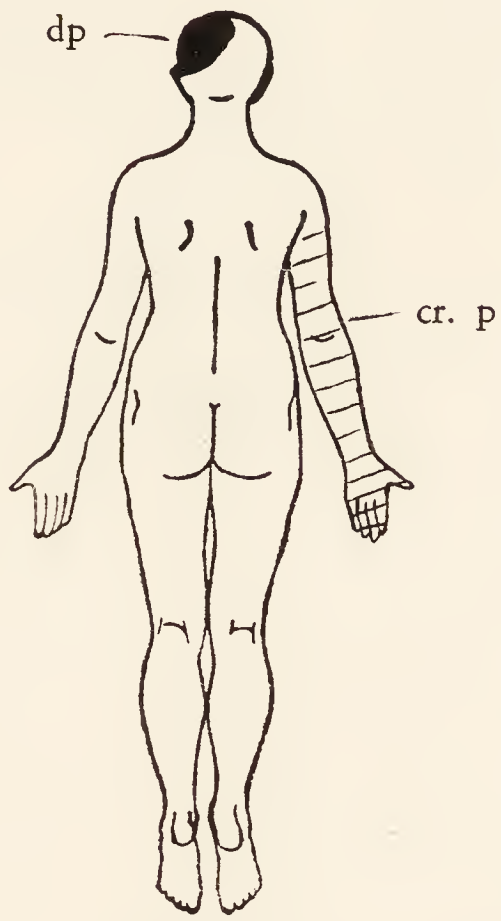

Fig. 18.
Leftsided meningeal affection accompanied by direct pains (dp) and paraesthesias localised to the upper extremity of the opposite side (cr. p).

as to what part of the trunk is affected. If, on the other hand, there is also in some part of the trunk a localised pain, it must be regarded as predominantly probable that both forms of pain are due to an affection of the nerve trunk on a level with the local pain, for pains emanating from the nerve sheath are of a direct kind and will thus indicate the level of the affection.

Similarly unilateral pains in the head accompanied by paraesthesias in the opposite side of the body, will indicate the probable site of the underlying lesion (fig. 18), and as

regards direct pains in combination with referred pains, direct pains due for instance to a local peritonitis proceeding from a lesion of flexura coli sin. will be localised to the left hypochondrium, while the referred pains emanating from the same section of the intestine will be localised to the infraumbilical reflex zone of the colon. (Fig. 19).

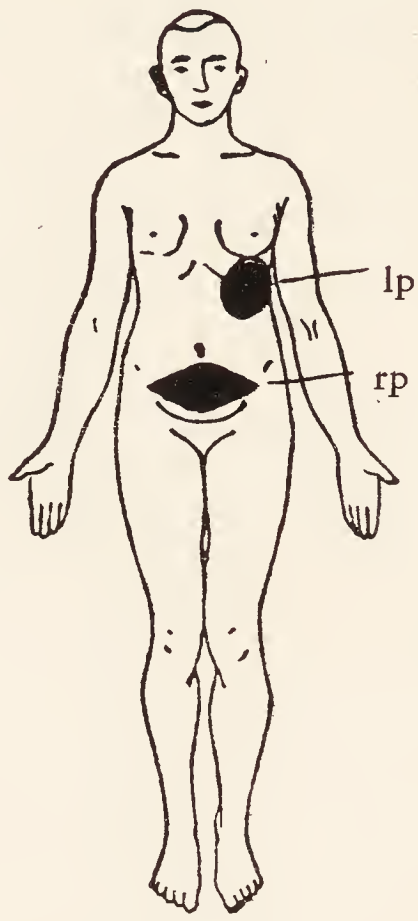

Fig. 19.
Affection of flexura coli sin. accompanied by local pains (lp) and by reflex pains (rp) localised to the infraumbilical zone.

Adherence, too, between the peritoneum and an underlying organ may cause direct peritoneal pain owing to tightening. If at the same time referred pains emanate from the organ in question because of the formation of stenosis, all the pains must again be regarded as combined pains. This is also the case when malignant organic diseases, by infiltrating the surrounding tissue, give rise to local direct pain while at the same time referred pains are evoked which are localised to the corresponding reflex zone. In figs. 20—23 are shown, in very diagrammatic

form, a series of cases of organic affections at the same time accompanied by direct local pain and indirect projected or referred pains.

In spite of the fact that the combined pains are not, theoretically, of any great interest, since they are combined of

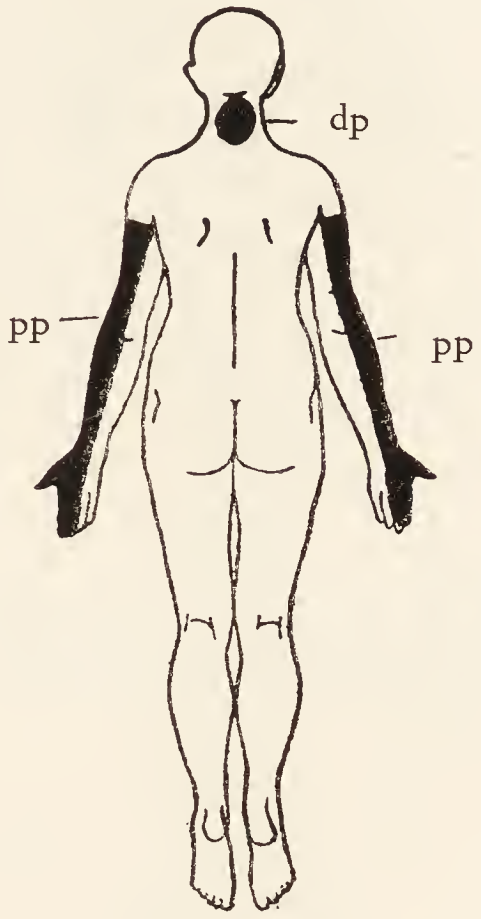

Fig. 20.
Lesion of v. c. V. accompanied by direct pains (dp) and projected pains (pp).

direct and indirect pains whose peculiarities have already been mentioned in the preceding lectures, it is necessary to keep in mind for practical diagnostical reasons that direct pains often accompany lesions of tissue elements which otherwise, when in a state of irritation, evoke projected or referred pains. An essential reason why referred pains especially are utilised less than desirable in diagnostics is presumably that clinicians have not ventured to pay attention to referred pains to such a degree as is done for instance by Mackenzie, because they know by experience that local pains, too, are frequently present. And as a matter of fact, there are clinicians who for that reason

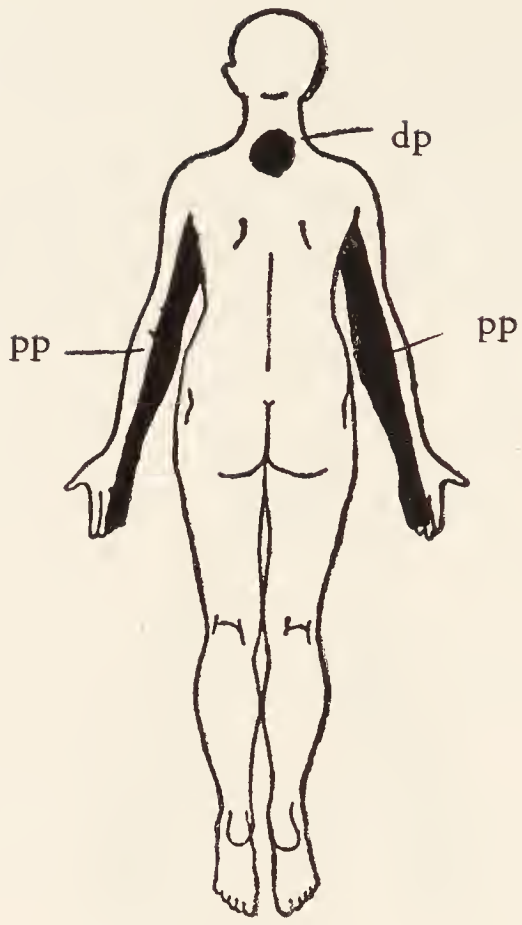

Fig. 21.
Lesion of VII v. c. accompanied by direct pains (dp) and projected pains (pp).

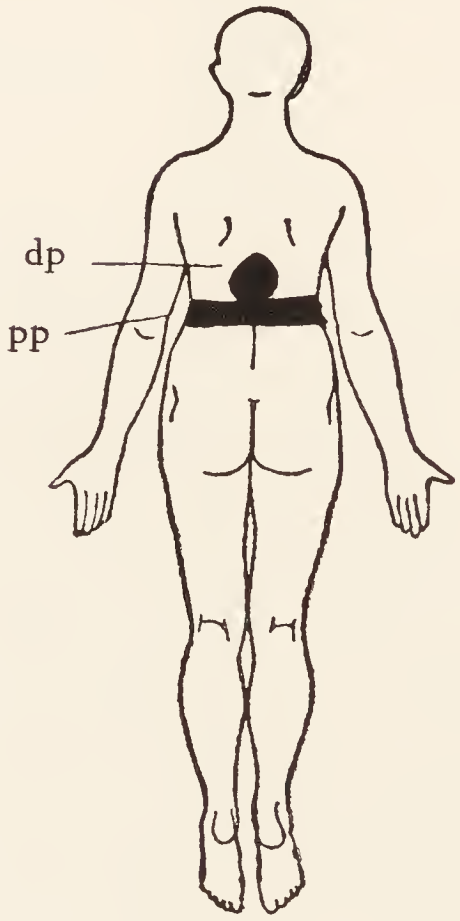

Fig. 22.
Lesion of v. d. X accompanied by direct pains (dp) and projected pains (pp).

entirely deny that the visceral pains must be regarded chiefly as referred pains. To such clinicians I would point out that pains accompanying visceral lesions are not to be regarded as an »either — or», but as a »both — and«, since there can hardly be any doubt that pains emanating from the viscera must

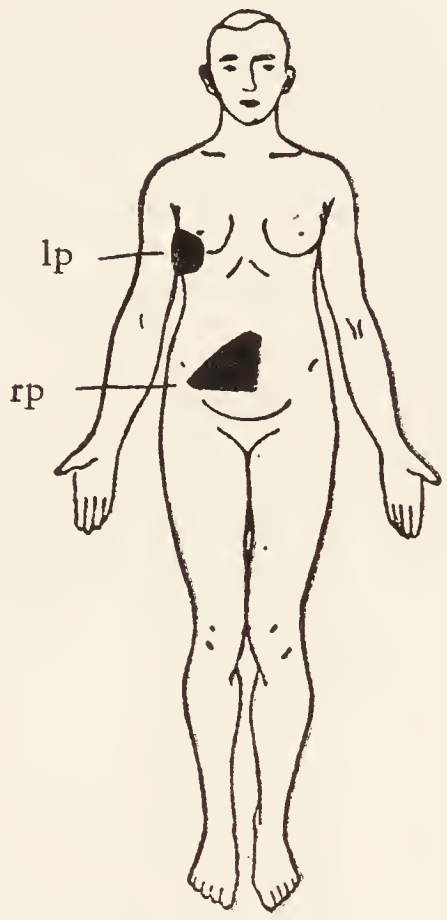

Fig. 23.
Rightsided pneumonia accompanied by local pleuritis pains (lp) and by appendicitis-like referred pains (rp).

chiefly be regarded as referred pains, but neither can there be any doubt that an organic lesion, by affecting the surrounding tissue, may in many cases give rise to direct local pains.

As a rule the investigator will not for any length of time be dependent for his diagnosis of a lesion exclusively on the patient's indication of pain. Sooner or later objective symptoms usually appear. But if for a long time pains should be the only symptoms, the investigator is not reduced to base his conclusions solely on the localisation and distribution of the pains. It is of vital importance to ascertain under what circumstances the pains occur, whether in connection with meals, defecation,

urination, menstruation etc. — The cause evoking pain, however, varies according as the pain emanates from one or the other organ or tissue, hence the facts coming within this field will be dealt with in more detail in the special part.

This brings to a conclusion my lectures on the general diagnostics of pain. In a later, larger work intended to form the foundation of a special diagnostic of pain I purpose to divide all pains occurring in man into 1) pains of the upper extremities, 2) pains of the lower extremities, 3) pains localised to the neck, 4) the back, 5) the chest, 6) the abdomen, 7) pains in the head, 8) pains in the face, and to analyse these pains. I shall then describe separately the clinically important types of pain and their differential diagnosis. —

While, in the present work, I have tried to throw light, in a brief and summary form, on the laws and rules governing the localisation and distribution of pain, I intend in the special part to describe at length the various clinically important forms of pain. The special part will contain references to the works of a considerable number of investigators besides those already mentioned, so the complete list of literature will be given in this special part of the diagnostics of pain. —

1a

1b